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NEW YORK CITY

IN TWENTY VOLUMES

VOLUME X

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DISEASES OF THE BRAIN.

BY

JOSEPH COLLINS,

NEW YORK.

DISEASES OF THE BRAIN.

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INTRODUCTION.

THE close of the nineteenth century furnishes an exceptional opportunity for a retrospective glance at the advances that have been made in the knowledge of the structure and architecture of the brain, in the interpretation of its physiological action, and in the application of the principles of modern pathogenesis to a solution of its manifold diseases. Contrasted even with fifty years ago, the advances that have been made are astounding. At that time Watson's "Practice of Physic" was a storehouse of up-to-date medical lore in the English language. To-day it is held in esteem not only because it is a historical repository, but because of the beauty of its style and the felicity of its clinical descriptions. In the light of our present knowledge, its comments on the causation of disease are lamentable, its teachings on pathogenesis absurd, and its therapy semibarbarous. It may be said that, withal, its clinical descriptions are unsurpassed, and this is readily granted. But descriptions of disease do not materially advance our ability to prevent or to treat them. Such ability is commensurate with progress in determining their causation, and the advances that have latterly been made in the conception, the localization, and the pathogenesis of intracranial disease have materially aided the prevention and cure of some of the conditions.

The architecture and the physiology of the brain are to-day, thanks to neurologists, histologists, and the school of experimentists, moderately well understood. It is, however, the application of the cardinal principles of pathology and pathogenesis, and particularly a comprehension of the relationship that exists between infection and the production of inflammatory diseases, that have been of importance in aiding us to discover the causes of some of these diseases, and to devise modes of diagnosis.

The intracranial diseases that have been placed on a substantial anatomical foundation during the past few years are encephalitis, including abscess of the brain, leptomeningitis, and intracranial new growths. With the understanding of their causation have come new means of diagnosis, and it is confidently hoped that these will be fol-

lowed by new methods of treatment, more successful than those now to hand. As an individual illustration of recent diagnostic measures of value, we may cite that of lumbar puncture of the subarachnoid space, a procedure the practical application of which we owe to Quincke, of Kiel. Without being oversanguine of the value of this measure, it may be said that it bids fair to become of great diagnostic importance in the recognition of more than one intracranial disease. Acute inflammatory affections of the brain and of its coverings, dependent upon similar causes, such as bacteria, are not materially different in onset and in the beginning of their clinical course, and every physician knows how difficult it is to make the differential diagnosis; yet, if the diagnosis can be made early, the prospect of the patient's recovery is materially increased, as there is then time for the proper application of remedies which may, in this early stage, abbreviate the tenure of the disease. I would not be understood as saying that the end of the nineteenth century sees us in possession of such measures, but the trend of pathological discovery at the present day is particularly in that direction.

The separation and recognition of primary and secondary degenerations have constituted a very important step in advance. The disease, disseminated sclerosis, may be cited as an instance. Formerly it was thought that this disease was a degenerative affection of the central nervous system, occurring in adult life and without attributable cause. To-day it would seem to be moderately well established that disseminated sclerosis is a disease of very early life, and that the islets of degeneration of the nervous system, which constitute its anatomical basis, are an expression of reaction to some hæmic irritant, such as a toxin which follows after the acute infectious diseases, like measles, scarlet fever, enteric fever, and, possibly, malaria. In the beginning such changes may be acute, or they may be very slow; but in the great majority of cases there is evidence to show that the degenerative process in the nervous tissue is mediated directly through the blood-vessels and that the process is a degenerative one from the start. It is surprising to think that pathologists have been unwilling to admit that the tissues of the brain, the parenchyma, let us say, may undergo degeneration—the same, for instance, as the tissues of the liver—without ever having been preceded by inflammatory change or accompanied by any of the concomitants of inflammation; but a study of the pathology of diseases of the brain to-day demands the necessity of such admission.

The birth of the neuron theory of the architecture of the brain has been accompanied by a flood of light in the interpretation, not only of the physiology of the brain, but of its pathology as well. It has

been suggested by some that a reconciliation of the claims of modern histology, concerning the anatomical and physiological unit, the neuron, and the facts on which the doctrine of cerebral localization is established, is impossible; but a moment's consideration will serve to show the untenability of such a view. It is not in place here to consider the details of the neuron—they are spoken of later on, and a familiarity with the history of the neuron on the part of the reader is taken for granted. It must needs be considered that the gray substance of the convolutions in the higher evolutionary stages is the essential organ of psychical elaborations and the seat of those functionings operative in the network that is formed by the terminal ramifications of the polymorphous cells of the cortex. This network, which is both the origin and the receiving station of all outgoing and incoming stimuli, is in connection with the periphery by the pyramidal cells which conserve sensory images, which images are the first materials of all intellectual light. The formation and preservation of motor and sensory images, their association and correlation, are the final products, and as all reaction to the environment is accompanied by motion in some form, these cells with their prolongations, protoplasmic and dendritic, represent the histological element on which the physiology and pathology of the brain are best and most logically explained.

In the section on localization it is shown that in different lobes of the brain are formed and preserved different images—in the central lobes the motor images, in the occipital the visual, in the temporal the acoustic, etc. Assumption of the neurons as the direct connection of each of these parts with their respective peripheral organs, and of the interrelation that exists between each of these functions by means of the dendrites and association tracts, is a necessity as well as a material and logical advance.

We are nearing the time, if, indeed, it be not already at hand, when the nosology of disease shall be built upon or in unison with the neuron theory, and already some workers in the science of neurology have heralded the advent of such classification. In the present consideration of intracranial diseases, the writer has preferred, however, to abide, as far as possible, by the classification based upon etiology, as he is convinced that such is more serviceable and intelligible to the practising physician than is any other.

No definite rules of diagnosis can be formulated to aid the physician in the recognition of diseases of the nervous system other than those in keeping with the golden rules of general diagnosis. In the domain of neurology, more depends upon routine, methodical, painstaking examination of the various components of the nervous system

in leading to the proper diagnosis than in any other department of medicine. If the physician will but have the patience to make such examination, and if when it is made he is possessed of the requisite understanding of the physiology of the nervous system and of ability to reason logically, it will not occur often that he is unable to make as accurate a diagnosis as the specialist of these diseases. Without these factors, however, no symptom complex in the domain of pathology can seem more complicated than that of an ordinary problem in neuropathology. It is not proposed in this connection to detail specifically the various methods by which a diagnosis of brain disease may be led up to. It should be said, however, that the examination of a patient suffering from intracranial disease does not differ materially from the examination of a patient suffering from any form of nervous disease. The point that the writer wishes to emphasize is that in every case the examination directed especially to the nervous system should be preceded or supplemented by an examination of every system of the patient's body. Not infrequently sufficient information is obtained from such general examination to put proper interpretation upon symptoms otherwise very obscure. I need only cite as an example the necessity of studying the pulse and the general vascular system in patients with brain abscess. Oftentimes information thus obtained may be the means of materially contributing to the patient's repossession of health by suggesting the application of proper remedies—such as, for instances, the detection of arterial capillary fibrosis in patients threatened with cerebral apoplexy.

The objective examination of the patient is of immense importance and the results of it are to be combined with information obtained subjectively; but the former is immeasurably more valuable in contributing to a diagnosis than is the latter. At a glance the physician may obtain from the countenance and appearance of the patient information concerning his feelings, states of mental content, suffering, and nutrition, which no amount of denial or affirmation on the part of the patient can materially change. Methodical examination to determine the integrity of motion, whether it be voluntary, involuntary, or reflex; of station, of coördination, of sensation, of vasomotor and trophic functions; of the complex of conditions, designated by the term intellection, should be made in every case, and deviations from the normal carefully noted, afterwards analyzed and synthetized. The varied abnormal manifestations of these, added to the constitutional symptoms of inflammation, go to make up the symptomatic content of all intracranial diseases. Their association and combination point to individual diseases. It is not considered necessary to enumerate and discuss the various forms of disturbance

of voluntary, involuntary, and reflex movements—such as tremor, convulsions, clonus, paradoxical contractions, spasms, contracture, automatic movements, dance movements, and the like that may occur with brain disease. It is believed that space devoted to such explanation can be more profitably employed. It is hoped, however, that the reader will bear in mind that diagnostic ability goes hand-in-hand with a full understanding and an intelligent recognition of these as well as of other perversions of brain function.

It is necessary to say a word in regard to some of the diseases which are here considered, and also to devote a few paragraphs to other alleged diseases that are not mentioned. Of the latter, anæmia and hyperæmia of the brain are given no space whatsoever. In a number of years devoted to the practice of medicine, the writer has never been called upon or impelled to make the diagnosis of cerebral anæmia or of hyperæmia as individual clinical entities, nor has he ever heard such diagnoses made by others of greater skill and wider experience with whom he has had the fortune to be associated. It is believed, therefore, that if these conditions occur they are most uncommon. Theoretically, hyperæmia of the brain is not an impossible condition; but practically its occurrence, independent of other intracranial conditions or general vascular derangements, is not common enough to warrant us in giving it separate description. The physician and the pathologist both recognize the occurrence of pulmonary congestion, of renal congestion, and of congestion of the liver; there can be no doubt that these conditions occasionally occur, and reasoning from analogy congestion of the brain ought to and possibly does occur. But congestion of the brain, compared with these even, is, it is believed, extremely rare. Not many years ago flimsy superstructures of symptoms were built, particularly by American and German writers, one to represent anæmia and the other hyperæmia of the brain. Nearly all functional nervous diseases, leaving out epilepsy, could be placed under the one or the other. For a time, happily a short one, these teachings were accepted by the profession, and long-drawn-out transcriptions of imagery, purporting to be a descriptions of anæmia and hyperæmia of the brain, found their way into many text-books and treatises. Later years have seen the space allotted to them materially curtailed, and that just in proportion as the neuroses and psychoses have become more universally recognized and understood; so that to-day, while admitting that cerebral hyperæmia may occur during a paroxysm of pertussis, or associated with a dilated right heart, or even as the result of intense mental application, particularly in connection with the ingestion of cardiac stimulants, and that anæmia of the brain may occur as a manifestation of general

hydræmia, the writer does not concede the necessity of considering these conditions as clinical entities nor apart from the factors that produce them.

Malformations of every kind and protal defects of the brain are considered very briefly, because these conditions excite a pathological interest rather than a clinical. Diseases of the brain and its coverings, due to parasites, are considered as briefly as possible, because in this country the rarity of their occurrence does not call for more extensive description. On the other hand, consideration of disease due to infection has been as detailed as space would permit, and the endeavor has been made to incorporate all that has so far been definitely proven in regard to the origin of such diseases, and this because of the conviction that satisfactory treatment will be forthcoming only when such causation is definitely and clearly made out.

In the section on cerebral localization scant mention is made of one of the most important pathological questions, and the most important psychological problem that can present itself to the physician, viz., aphasia, and this because the subject is considered in this treatise apart from cerebral localization and by another writer.

Current medical literature, and not previous treatises and textbooks, has been the source where information has been sought, and the endeavor has been made to express obligations to such sources by appending to each section a register of the literature that has been consulted.

MORPHOLOGY AND ANATOMY OF THE BRAIN.

In the preparation of this section on the morphology and anatomy of the brain, it has been my endeavor to keep in mind that it is intended more for the general practitioner than for him who has become so familiar with the anatomy and diseases of the nervous system as to warrant his assumption of the name of neurologist. The anatomy of the central nervous system, more particularly the part within the skull, requires closer study and deeper thought for its comprehension and understanding than does any other subject with which the student physician has to cope.

A complete understanding of the intricacies of its architecture is the labor of a lifetime. A knowledge of its anatomy sufficient to comprehend the problems of those normal and diseased functionings of the brain which the physician is liable to meet every day is more easily acquired. An outline sufficient for the practical needs of the physician will be attempted here.

This outline will comprise: 1. The gross anatomy and morphol-

ogy of the brain, with particular reference to the external conformation of the cerebral hemispheres. 2. The finer structure in so far as this is necessary for the comprehension of the pathways of receptive stimuli or the sensory tracts, and of emissive stimuli or the motor tracts. 3. The circulation of the brain.

The physiology of the brain will be considered with particular reference to localization of function.

The physician who is not versed in the nomenclature of the fine and gross anatomy of the nervous system is likely to be confused by the great mass of, to him, unnecessary and unessential detail. Another obstacle to the general reader in the way of an understanding of the central nervous system is the entirely different nomenclature which different writers employ in the designation and description of well-known parts of the brain, particularly of the cerebrum. I shall endeavor to avoid this obscuration as much as possible by the use of the most universally accepted terminology and by sometimes adding in parenthesis immediately following it the most commonly used technical synonym or favorite term.

The terminology proposed by Wilder and adopted by his students, by Mills, and in part by other writers, I shall make but little use of, because I have not been able to convince myself that it contributes to lucidity.

The brain (encephalon) is divided embryologically into five parts: (1) The cerebrum which includes the hemispheres, the striate bodies, the corpus callosum, outer commissure, and fornix; (2) an interbrain, including the structures in connection with the third ventricle, principally the optic thalami, the median commissure, the mammillary bodies, and infundibulum; (3) the midbrain, comprising the crura cerebri, the corpora quadrigemina, the optic thalami with the middle commissure and pineal body; (4) the hindbrain, made up of the cerebellum with its peduncles and the pons (pons Varolii), and (5) the afterbrain, the oblongata (medulla oblongata) or bulb.

The cerebrum, a large ovoid mass, consists of two equal parts, the hemispheres (hemicerebrums), each containing a large cavity the lateral ventricle, united together on the mesal surfaces by a large white mass of fibre known as the corpus callosum. The two hemispheres assist to form another cavity, the third ventricle. The hemispheres are separated by the longitudinal fissure.

One hemisphere is the exact counterpart of the other and has an anterior or frontal pole, a posterior or occipital pole, three surfaces, an inner (mesal), which, with its fellow of the opposite side, forms the lateral borders or sides of the longitudinal fissure, an outer surface, having a superior and lateral aspect, conforming to the slope of

the skull, and an under surface which is divided into three parts to conform to the three fossæ of the skull with which it comes in contact: the orbital, the middle, and the occipital.

A gross division of each surface of a hemisphere is often made into lobes. The names which are given to these on the external surface, including the upper, outer, and under surfaces, are the frontal, parietal, occipital, temporal, and insular lobes (the Island of Reil), the names of which designate in a general way their location, with the exception of the last, which is covered over by the Sylvian fissural border of the temporal and parietal lobes. The insular lobe is internal, but external and adjacent to the cerebral ganglia. In the early formative stages of the brain it shows first on the external surface, being covered over gradually as the brain develops. On the mesal surface of the brain the same lobes, with the exception of the insular, are seen; but here special names, which will be mentioned later, are given to some of them.

Like the entire central nervous system, the cerebrum is made up of two parts which from their appearance are known as the gray and the white matter. The former, situated on the outside, is convolutive, that is, it dips into the numberless fissures which apparently break up the continuity of the surface of the brain. The entire mass of gray matter makes up the cortex of the brain. By means of this involution of the gray matter into the fissures, its continuity is unbroken, and its surface area is much greater than that of the white which it covers and encloses. It is calculated by Wagner that the extent of the sunken surface of the gray matter is twice that of the exposed. The thickness of the cortex varies in different portions of the brain and varies according to race, age, sex, and individual. The average thickness of the cortex is from 1.5 to 3.5 mm.: it is thicker at the crest of the convolution than at the bottom of a fissure; it is thicker in the central lobes than anywhere else in the brain, and thinnest in the occipital lobe. The cortical area is somewhat greater in the brains of highly intellectual individuals than in those of ordinary mental capacity, and the investigation of Donaldson seems to show that brain regions developmentally imperfect, owing to absence or defect of function, are deficient not only in cortex thickness but in cortical surface area.

The landmarks of the hemispheres are the fissures (sulci). They are usually spoken of as of two kinds: the principal, "interlobar" or complete fissures, and the secondary, "interlobular," "interconvolutional" or partial fissures. The important fissures on the outer surface of the brain are the fissure of Sylvius, the fissure of Rolando (central fissure), and the parietooccipital fissure. The principal fissures on the mesal surface of the brain are the calcarine, which con-

tinues in part to the parietooccipital fissure of the outer surface, the callosomarginal fissure, and the collateral fissure.

The fissure of Sylvius, the largest and most striking of the fissures, is on the under and lateral surfaces of the brain. It begins as a rather deep fossa at the anterior perforated space, passes forwards and upwards to the lateral surface of the brain, where it divides into a short anterior horizontal branch and an ascending branch which continues backwards obliquely towards the longitudinal fissure and terminates in a bifurcation near the middle of the lateral surface. When the lips of the fissure are pulled apart just above its origin the insular lobe (island of Reil) comes into view. The fissure of Sylvius, when it turns over upon the lateral surface of the brain, forms a flat triangular depression, which is often called the fossa of Sylvius, and into which the frontal and parietal convolutions reach like a cover over the island of Reil. For this reason they are oftentimes referred to as the opercula or, if spoken of individually, they comprise the frontal operculum, the parietal operculum, and the temporal operculum. When the fissure appears on the lateral surface of the brain it separates the frontal and parietal lobes from the temporal. Just behind a point midway between the anterior and the posterior poles of the brain, on the lateral surface of the brain, is the deep, conspicuous fissure of Rolando (central fissure), which on account of the fact that it nearly evenly divides the motor areas of the brain is of great importance in motor localization. Its course is obliquely upwards and backwards from its origin near the fissure of Sylvius, and it terminates just before reaching the longitudinal fissure. Sometimes it is confluent above with the longitudinal fissure, but more frequently it is not. It separates the frontal from the parietal convolution. The parallel convolutions on both sides are known as the ascending frontal and the ascending parietal convolutions, frequently and properly merely as the central convolutions.

The parietooccipital fissure can be seen for a short extent only on the external surface of the brain. It separates the parietal from the occipital lobe. On the mesal surface it unites below with the calcarine fissure; the wedge-shaped portion of the occipital lobe included between these two fissures is called the cuneus.

The callosomarginal fissure is the most conspicuous fissure on the mesal surface of the brain. It runs parallel to the corpus callosum as far as the middle of the hemisphere, where it turns obliquely towards the upper border of the hemisphere, terminating just posterior to the beginning of the fissure of Rolando. It separates the gyrus fornicatus, forming its upper boundary, from the frontal convolution anteriorly and from the paracentral convolution posteriorly,

forming the posterior boundary of the latter and separating it from the precuneus. In the latter location the fissure is often referred to as the paracentral fissure.

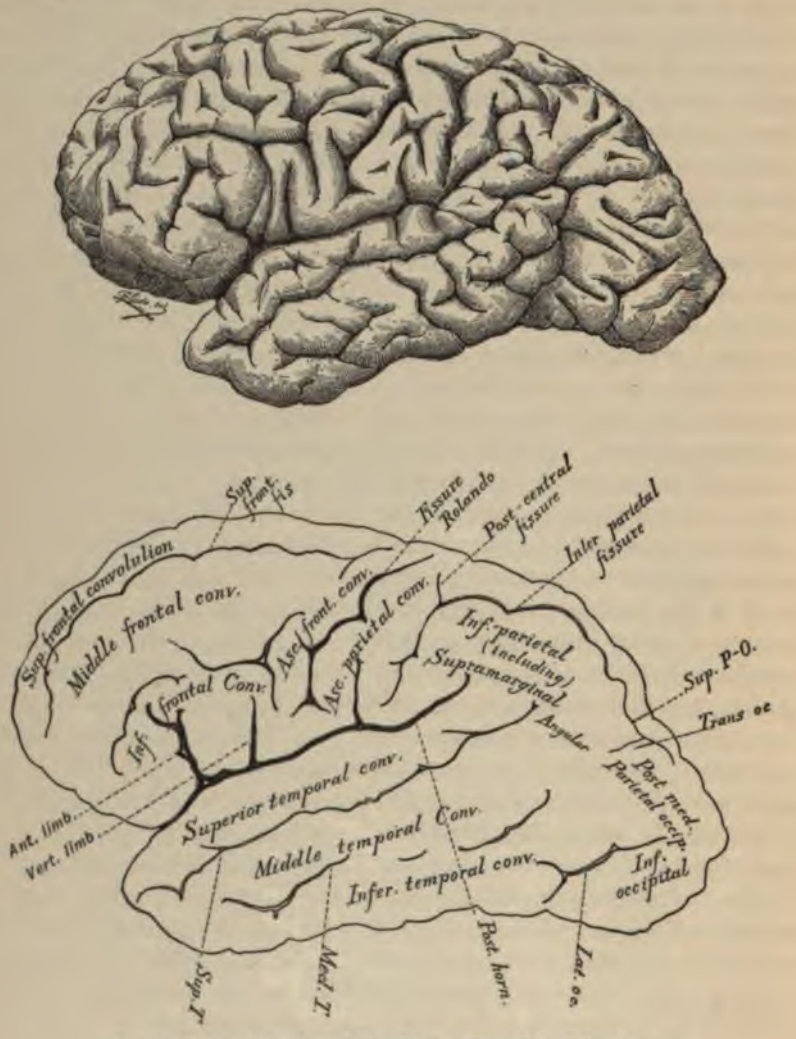

Fig. 1.—External Surface of the Brain. From Van Gehuchten.

The calcarine fissure is a deep fissure cleaving the mesal surface of the posterior pole of the brain, and is composed of two portions: an anterior limb which separates the precuneus above from the temporal lobe below, extending forwards and terminating just beneath the splenium of the corpus callosum; and a posterior limb, which

separates the cuneus from that portion of the temporal lobe known as the lingulum and which reaches the anterior apex of the occipital pole of the hemisphere.

The collateral fissure first described by Huxley and recognized by Schäfer lies below and parallel to the anterior part of the calcarine. It extends forward towards the tip of the temporal lobe but without reaching the extremity of the lobe.

As has been said, these fissures just enumerated divide the brain into parts, so-called lobes, which, on the external surface, have received names similar to the cranial bones which cover them—for example, the frontal, parietal, temporal, and occipital. In a general way all of the brain in front of the fissure of Rolando and above the fissure of Sylvius is comprised in the frontal lobe. The part of the brain behind the fissure of Rolando above the fissure of Sylvius and in front of the parietooccipital fissure constitutes the parietal lobes; that behind the parietooccipital fissure and above the posterior limb of the calcarine fissure is occipital, while that below the fissure of Sylvius in front and the anterior calcarine fissure mesally is included in the temporal lobes. The boundaries of the insular convolution have already been considered. The distinctions between the various parts of the brain are by no means so striking and apparent as might seem from this arbitrary way of bounding them. Attempts have been made to divide the surfaces of the brain according to their physiological functions, and the trend of embryological and pathological investigation is decidedly in this direction. At present division into motor, sensory, special sense, and pyschical area, such as are spoken of under "cerebral localization," are justified. The mesal surface of the brain is divided between the lobes mentioned on the external surface. Special names have been given to different convolutions on this surface on account of their shape, and their fissural and boundary relation.

To readily grasp the problems of localization it is necessary to consider the convolutions of the different lobes in detail, particularly those in which there is accurate localization of function. It may be said in passing, however, that verbal description is as much inferior in worth to faithfully executed drawings as drawings are to the actual brain itself. It is therefore recommended that the accompanying illustrations (Figs. 1, 2, 3, and 4) be carefully studied.

The frontal lobes, which comprise about one-third of each hemisphere, are composed of four gyri (convolutions): on the external surface (Fig. 1) the first, second, third, and ascending frontal. On the mesal surface (Fig. 2) the frontal lobes are represented by the marginal gyrus; on the inferior or orbital surface (Fig. 3) by the orbital gyri,

outer, middle, and inner, which latter is known as the gyrus rectus, and a small gyrus behind near the insular convolution known as the posterior orbital. The principal fissures of the frontal convolutions are the precentral, which runs parallel with the central or fissure of Rolando, and separates the ascending frontal behind from the superior

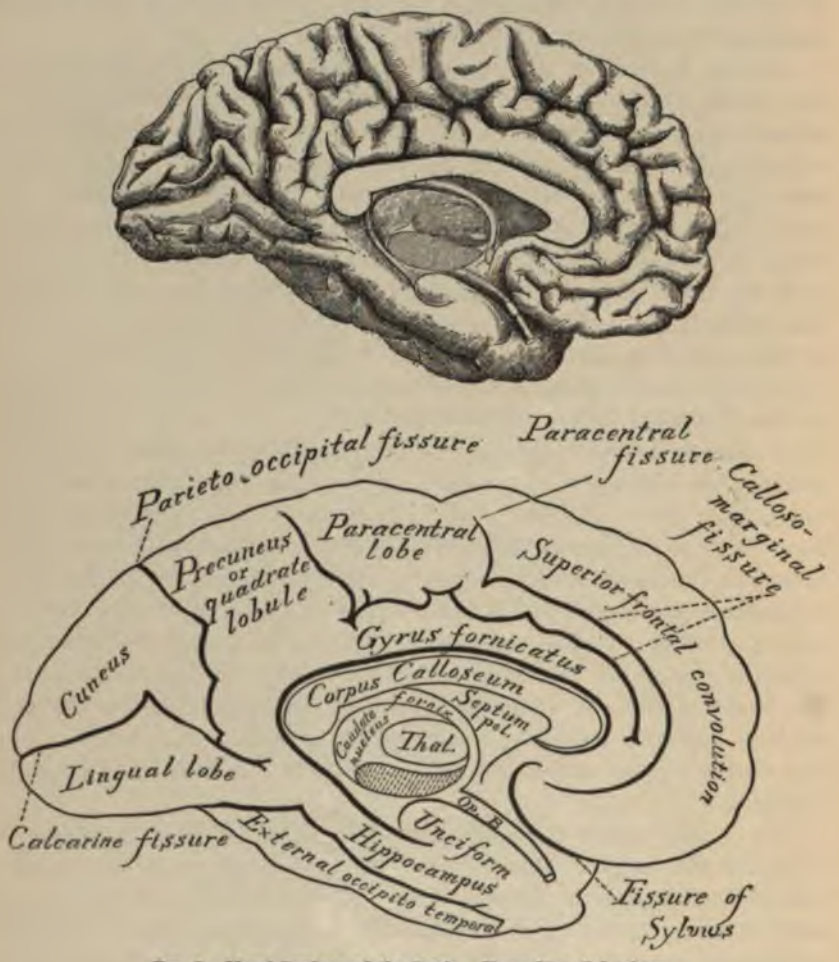

FIG. 2.—Mesal Surface of the Brain. From Van Gehuchten.

and middle frontal convolutions anteriorly, and the superior and inferior frontal fissures between the first and second and the second and third frontal gyri respectively.

The boundaries of the parietal lobe have already been considered. The important fissure which it contains is the interparietal. This begins at the anterior inferior angle of the parietal lobe, just

behind the fissure of Sylvius, and passes first upwards, nearly parallel to the fissure of Rolando, and then backwards to the end of the parietal lobe almost to the parietooccipital fissure. It divides

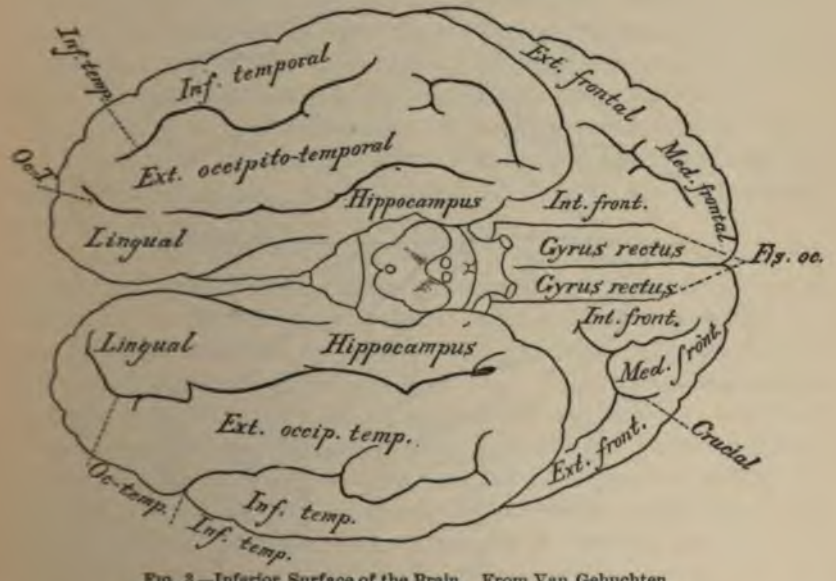

FIG. 3.—Inferior Surface of the Brain. From Van Gehuchten.

the parietal lobe posterior to the ascending parietal into two parts: the superior parietal which borders the longitudinal fissure, and the inferior parietal which is contiguous below with the temporal lobes. The ascending parietal convolution or postcentral convolution lies

immediately behind the fissure of Rolando and parallel with the ascending frontal convolution. The superior parietal lobule is sufficiently described by mention of its boundaries. The inferior parie-

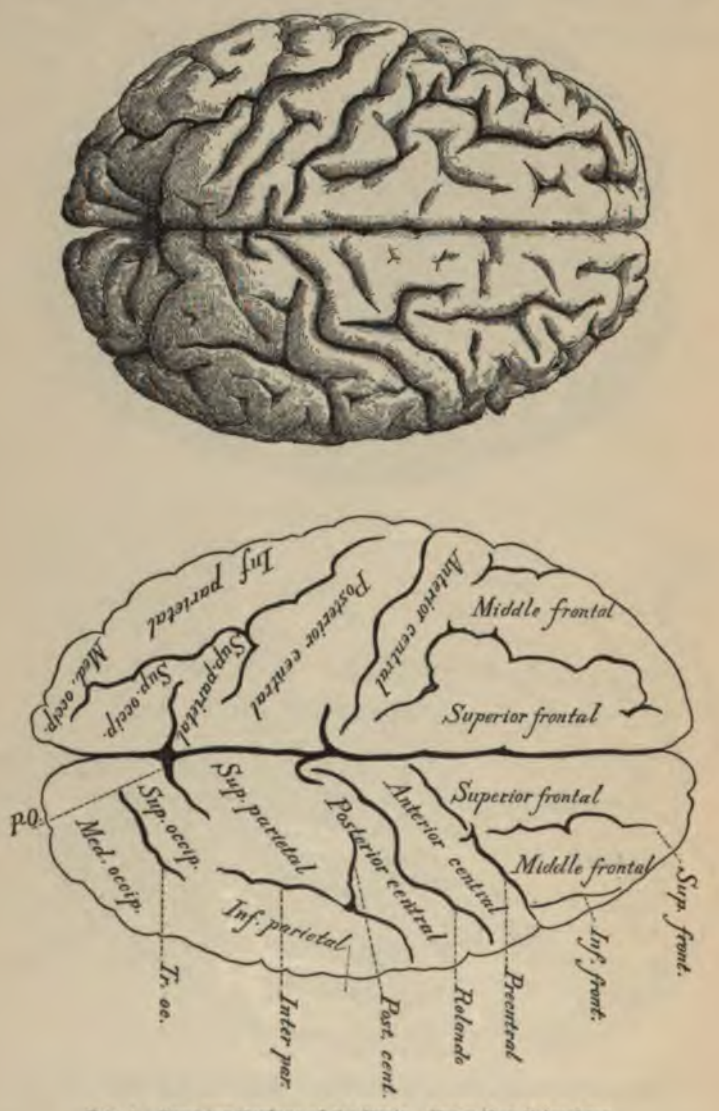

FIG. 4.—Superior Surface of the Brain. From Van Gehuchten.

tal lobule lies in the concavity of the interparietal sulcus and is divided into three gyri or convolutions, which enumerated from before backwards are the supramarginal, the angular, and the post-

parietal convolutions. As these three gyri are the battleground of serious controversy by certain localizationists it is well perhaps to describe them in somewhat greater detail than the remaining convolutions. The terms marginal, supramarginal, and inframarginal are rather loosely used by some writers. I shall use the term supramarginal gyrus to indicate the lobule bounded in front by the ascending parietal gyrus, above by the horizontal limb of the interparietal fissure, behind by the angular gyrus, and below by the supertemporal convolution, the latter sometimes called intramarginal—a horseshoe-shaped gyrus which embraces the posterior end of the fissure of Sylvius. (This supertemporal or inframarginal convolution is not recognized by all anatomists. It is looked upon as a part of the supramarginal gyrus.)

The term angular gyrus has been used with even greater latitude, particularly because of the less restricted definition of the area in the brain of the ape, the angular gyrus of Huxley, *pli courbe* of Gratiolet. By the majority of writers it is used to include all that remains of the inferior parietal convolution posterior to the supramarginal convolution, that is, the part of the parietal convolution between the superior parietal convolution above, the occipital behind, and the temporal below where these three parts come together. It will be seen that this outline follows Schäfer in limiting this area of the angular convolution by describing a postparietal posteriorly to it.

The parietal lobe is represented on the mesal surface of the brain by the precuneus or quadrate lobule; these two terms indicate its position and its shape. It is limited in front by the paracentral fissure and behind by the parietooccipital.

The occipital lobe forms the occipital or posterior pole of the hemisphere. A knowledge of its boundaries and relationships is of extreme importance to the physician because in it is localized the sense of sight. On the external surface it is not distinctly demarcated from the parietal and temporal lobes. This surface is confined to comparatively small limits by Eberstaller and Schäfer, who give as boundaries on the external surface, in front the parietooccipital and the anterior occipital fissures, and below a line connecting the lower end of the anterior fissure with the posterior extremity of the calcarine fissure. On the mesal surface, its limitations are very distinct in front by the parietooccipital fissure and below by the calcarine; the resulting gyrus, triangular in shape, is known as the cuneus, or cuneate lobule.

The remainder of the hemisphere to be seen on the external surface is comprised in the temporal lobe, which is separated in front and above from the frontal and parietal lobes by the fissure of Syl-

vius; behind, the delimitation from the parietal lobe is not distinct, while still farther posteriorly it is separated from the occipital lobe by the later occipital sulcus. The temporal lobe taken as a whole is kidney-shaped with the convexity directed outwards, its anterior narrower end projecting beneath the orbital surface of the frontal lobe and separated from it by the fossa of Sylvius. Its upper surface is roofed by the posterior limb of the fissure of Sylvius, while its lower surface is separated from the gyrus fornicatus (or limbic lobe as designated by Schäfer) by the collateral fissure. On the external surface the temporal lobe is separated by four longitudinal fissures, which divide it into five gyri which are called respectively from above downwards the first, second, third, fourth, and fifth temporal gyri. Simple enumeration of these gyri is sufficient for our purpose except in the case of the fourth and the fifth temporal. The first of the former convolutions is very commonly referred to as the fusiform lobule and is considered to be a part of the occipital lobe, while the fifth is usually spoken of as the lingual lobule. It is situated between the collateral and calcarine fissures and for this reason is often called the infracalcarine or subcalcarine gyrus. The island of Reil (insular convolution) is, as has been said, not perceptible on the surface of the brain until the covering parts of the frontal and parietal lobes (opercula) are pulled back or cut away. When this is done it will be seen that this lobe is somewhat pyramidal in shape, the base resting on the corpus striatum with which it develops, its apex projecting outwards towards the bottom of the fissure of Sylvius. Its principal fissure is the central fissure of the island, which running parallel to the fissure of Rolando separates it with other less conspicuous fissures into several straight convolutions, gyri recti.

The inner surfaces of the hemispheres—the surfaces which are united by means of the corpus callosum—have been described in so far as the external lobes of the cerebrum are there presented. There remains, however, a portion which it is necessary to mention specially in order to obtain a clear idea of its location. This portion was named by Broca the *grande lobe limbique* and is described by Schäfer as the limbic lobe. It is bounded above by the callosomarginal and paracentral fissures which separate it from the frontal and parietal lobes, below by the anterior part of the collateral fissure which separates it from the temporal lobes, behind by the postlimbic which separates it in part from the precuneus. Speaking generally, it arises from below and in front of the corpus callosum just above the olfactory tract and encircles the corpus callosum like a girdle; passing around behind and beneath the splenium of the latter, it continues anteriorly beneath the crusta as far forward as the olfac-

tory lobe, quite to the point of its beginning. From the point posteriorly where its continuity is interrupted by the entrance of the calcarine fissure, that is above the collateral fissure to its termination, it is known as the hippocampal or uncinate gyrus. This lobe is often referred to by writers as the falciform lobe, a name given to it by Schwalbe, but which included also the fascia dentata, fornix, and septum lucidum, structures which are to be seen only when the hemispheres are separated. Schäfer uses the limbic lobe to include all this and in addition such rudimentary gyri as the supracallosal, geniculi, and subcallosal—parts which do not interest us now, as they have no "localization" significance. The limbic lobe is made up of three gyri or convolutions, the gyrus fornicatus, the hippocampal gyrus, and a less important one, the dentate gyrus. The gyrus fornicatus, gyrus cinguli, or callosal gyrus, is immediately over the corpus callosum and separated from it by the fissure of the corpus callosum. It is frequently described as consisting of two portions, the gyrus cinguli, the part lying close to the corpus callosum, and the gyrus hippocampi. The gyrus fornicatus is separated, however, by a very conspicuous narrowing known as the isthmus of the gyrus fornicatus, and from this point forward to its termination near the apex of the temporal lobe it is properly known as the hippocampal gyrus. The gyrus hippocampus together with the infracalcarine gyrus is often spoken of as the uncinate gyrus on account of the hook-like shape of its terminal point known as the uncus.

The dentate gyrus is a small convolution with a serrated border just above the hippocampal gyrus, immediately dorsad of the splenium of the corpus callosum: it lies on the isthmus of the gyrus fornicatus and extends along the border of the hippocampal gyrus to the space between it and the uncus. It is well to remember that the dentate gyrus forms the surface of the posterior aperture of the lateral ventricle.

The olfactory lobe, like the optic, is rudimentary and was included by Broca as a part of the limbic lobe. It is composed of two portions, the posterior and the anterior olfactory lobules, the former immediately in front of the anterior termination of the gyrus hippocampus. From the latter arises the olfactory tract which extends forward to receive the olfactory nerves.

The Histological Structure of the Gray Matter.

The finer structure of the gray matter has been clearly demonstrated during the past few years, principally on account of the methods of metallic impregnation, the method of Golgi and S. R. Cajal,

Lenhossek, Van Gehuchten, Kölliker, and a great host of workers, and by the application of the methyl-blue method of Nissl.

The constituents of the brain are developed from the ectoderm and from the mesoderm; the neural elements and the neuroglia being ectodermal, the vascular elements mesodermal. The specific neural elements are the ganglion cells and the nerve fibres. The remainder, which constitutes the supporting substance or neuroglia, is divisible into ependyma cells, glia cells, ependyma prolongations, and glia prolongations. The mesodermal elements are the arteries, veins, capillaries and lymph vessels, and their constituents.

The importance of recognizing the unity of embryological origin of all the neural constituents is very great from a pathological standpoint. Considering the glia cells and ependyma cells apart from the specific neural elements as a so-called supporting framework for the parenchyma has done much to retard the proper histological and pathological appreciation of the significance of these elements.

The cortical gray matter has commonly been described as made up of six layers, judged by the older methods of hæmotoxylin and carmine staining. Neuroanatomists are now agreed to consider it as made up of four layers of cells, named respectively, from the periphery inwards, the superficial zone, the zone of small pyramids, the zone of large pyramids, and the zone of polymorphous cells (Figs. 5, 6). The superficial zone is made up of cells of four types, polygonal, fusiform, triangular, and unipolar—the first two being least numerous. Each fusiform cell gives off an axis-cylinder process from its apex and its base which run horizontally or diagonally for considerable distances, never, however, into the adjacent layer, and split up near the periphery. The collaterals of these processes form very rich plexuses of terminals.

The second zone, the layer of small pyramids, is composed chiefly of pyramidal cells which send the axis-cylinder processes given off from their apices into the superficial layer where they terminate in tufts. The collaterals of the cells and the axis-cylinder processes divide dichotomously. The expansions of the lateral twigs thus form for a considerable distance a broad band, the stripe of Baillarger.

The zone of large pyramids, beneath the layer just described, is universally considered to be the seat of origin of motorial impulses. The cells do not differ from those of the zone of small pyramids except in size and in the thickness of their processes. In shape they are polygonal, egg-shaped, spindle-shaped, and triangular. They vary in size and number in different parts of the brain and are largest and most numerous in the ascending frontal and ascending parietal con-

volutions. In the cortex of the frontal pole they are small and scarce and in the occipital pole they are absent. The prolongation of axis-cylinder processes forms, as we shall see later, the projection system of the first order, beginning with the corona radiata.

Fig. 5.

The network formed by the collaterals of their axis-cylinder processes constitutes what is known as the thin stripe of Baillarger. The striking feature of this layer is that the apical process does not reach into the superficial zone.

The fourth zone, the zone of polymorphous cells, is not yet well understood from the morphological point of view. The cells of this zone are, as their designation indicates, of different shapes, the principal kind being that described by Ramon y Cajal as the cells of short axis cylinder which occur here and also in very much smaller number in the second and third layer. Spindle-shaped cells and solitary cells have been described as constituents of this layer by Marinotti and Golgi respectively.

We have mentioned before that the specific neural elements are the

Fig. 6.

nerve cells and the nerves fibres: every nerve fibre is the prolongation of the axis cylinder (neuraxon) of a ganglion cell. Each nerve cell possesses a cell body, a nucleus with a nucleolus, and the so-called processes. The nucleus with its nucleolus occupies the central part of the cell; it is surrounded by the so-called protoplasm of the cell body. From the latter there go off sprouts, which are designated processes of the cells. By means of the Nissl methyl-blue stain, it can be demonstrated that the cell body is composed of two kinds of substances, which differ from each other in their reaction to this stain. One of these substances retains the blue stain in the differentiation fluid that is used; the other loses it, so that it appears colorless. We speak, therefore, of a chromatic and an achromatic part of the substance of the cell body.

The chromatic part is arranged either in the form of corpuscles, distributed so as to give the cell a large, spotted, tiger-hide-like appearance, or in the form of threads, chains of fine granules, etc.

The so-called achromatic part of the substance of the cell body has evidently a fibrillary structure, and this is probably continuous with the fibres of the so-called axis cylinder, of which we shall soon speak.

By the Nissl method the so-called processes of the cells are brought out, but they can be traced only for a short distance. It is seen that they also possess chromatic and achromatic substance; the former being usually represented in the shape of large spindle or pyramidal shaped corpuscles. One of the processes distinguishes itself from the others by its mode of origin. It is observed that part of the cell body from which this process originates is unstriated, being marked by an ear-shaped white area, which has a fibrillated appearance. This particular process is called the axis-cylinder process, or nervous process, while the others are called protoplasmic processes.

While Nissl's methods demonstrate the internal structure of the cell body, and in part the protoplasmic processes most exquisitely, the finer ramifications of the processes cannot be brought out with this stain. In the methods of metallic impregnation discovered by Golgi and modified by many investigators since that time, we have, however, the means at hand to bring out the finest ramifications in the clearest manner, and we are enabled to follow the delicate threads into which these processes divide for a very long distance, and to observe the manner of their distribution and relation to other elements.

Investigations with the methods of metallic impregnation have brought out the following facts: There are two kinds of processes—the protoplasmic processes or dendrites, and the axis-cylinder or nervous process. While the protoplasmic processes are usually multiple, there

is always only one nervous process. There is no doubt that the nervous process has the special function of conducting nerve impulses.

The functional part which the protoplasmic processes play is still disputed. Some authors (Golgi) ascribe to them nutritive functions exclusively; others believe that they also serve in the conduction of impulses (centripetally from the periphery towards the cell body). The nerve process is to be distinguished from the protoplasmic proc-

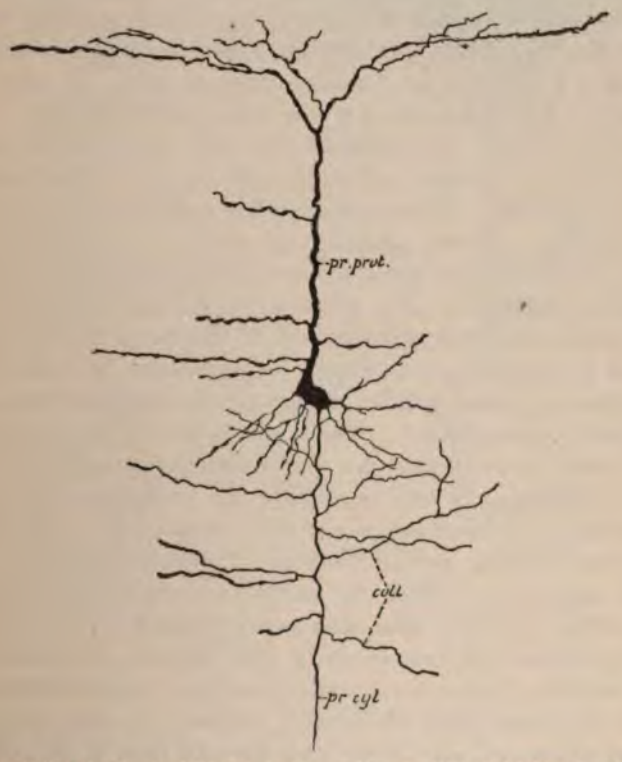

FIG. 7.—Nerve Cell of the Cerebral Cortex of a Young Mouse. (Van Gehuchten.) *Pr. prot.*, protoplasmic prolongation; *coll.*, collateral branches of the axis-cylinder prolongations; *pr. cyl.*, axis-cylinder prolongation.

esses by its extreme fineness. A glance at Fig. 7 will show the extreme richness and extent of the ramifications of the processes, and especially of the nerve processes. In regard to the structure and extent of the nervous processes, various types of cells have been distinguished. It will be seen from Fig. 8 that in some cells the nervous process divides itself up in numerous branches, which subdivide again and again, so as to form lattice-like formations or "arborizations." In other cells, again, the nervous process does not divide immediately

after leaving the cell; it can be followed as a continuous thread for a long distance. The teachings of neuroanatomists to-day are towards a treatment of the ganglion cell with its axis-cylinder prolongation as an anatomical and functional entity, to which the name neuron (Waldeyer) is given. Almost all ganglion cells have but one axis-cylinder prolongation, although some axis-cylinder prolongations divide into two. The course of this prolongation is generally a long one, and it gives off in the mean time but few lateral branches, the so-called collaterals which course for a greater or lesser distance in pericellular baskets or network (Fig. 8). At its termination, which is always around certain individual or some nest of ganglion cells, it splits up into terminal end branches, technically known as terminal arborizations. The ganglion cell has other prolongations than the one which is called the axis-cylinder prolongation; all these split up soon after their origin into a fine network, which are traceable only a short distance after their branching. These processes are known as protoplasmic prolongations in contradistinction to axis-cylinder prolongations, or more commonly as dendrites. The terminal branchings of these dendrites as well as the terminal arborization of the nerve fibres are always free—that is, there is no continuity or confluence between them and the dendrites of other cells or of the terminal arborizations of other axis-cylinder prolongations. Their connection is by simple contact only. It was formerly thought, and is still by some (Golgi, Daziel) that the ramifications and especially the end arborizations of the nerve processes form anastomoses with those of the other nerve elements, thus forming actual networks through which the impulse could be carried over from one nerve element to the other. It is almost certain, however, that each nerve element with all of its ramifications preserves an individuality and that the transmission of a nervous impulse from one element to another occurs without anastomosis of the anatomical elements. Contact or close proximity of the ramifications of one nerve element with that of the other subserves this function.

Neurons are divided first of all, according to their function, into motor and sensory. Each of these is divided into a primary or central neuron and a secondary or peripheral neuron. A primary motor neuron consists of a ganglion cell in the motor areas of the brain (ascending frontal and ascending parietal convolutions) and its axis-cylinder prolongation, which extends to ganglion cells in the oblongata and cord around which cells the arborization of the axis cylinder takes place. This constitutes the corticonuclear division of the motor tract. A secondary or peripheral motor neuron is made up of a ganglion cell around which the arborization of the axis-cylin-

der prolongation of the central neuron takes place, its axis-cylinder prolongation, and its terminal construction in muscles or glands. It constitutes the peripheral motor tract.

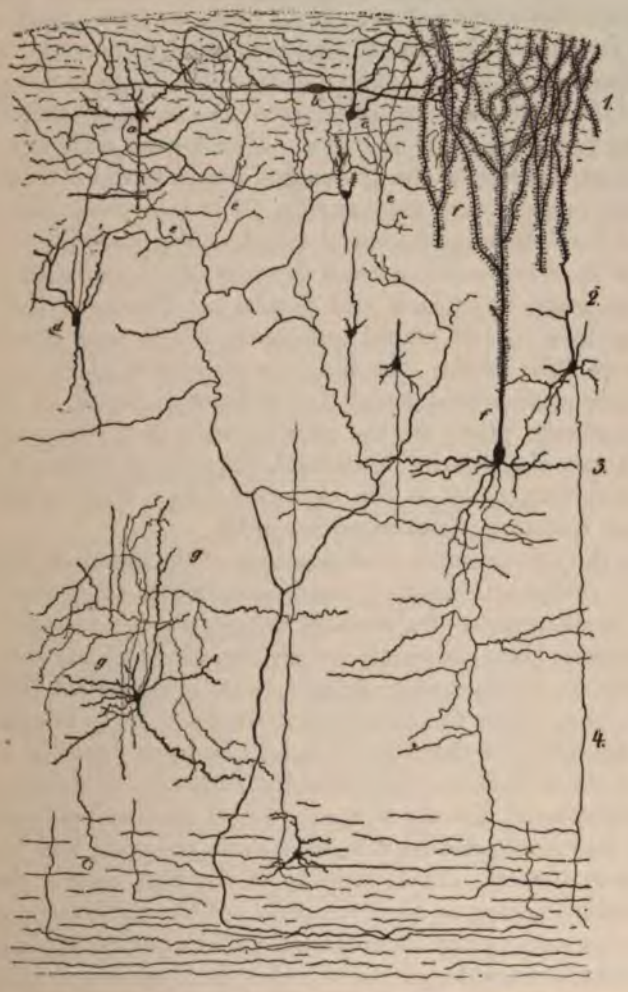

FIG. 8.—Section through a Mammalian Cerebral Cortex. Combined from preparations after S. R. Cajal. (Edinger.)

The sensory neuron has its primary ganglion cell, not within the central nervous system, but external to it in the spinal ganglia and possibly also in peripheral parts of the body. A cell of the spinal ganglia gives off an axis-cylinder process which divides into a large and a small fibre. The former goes to the periphery of the body, the

latter goes into the spinal cord, forming the posterior root. The course of the fibres that go to the periphery of the body needs no description. The fibres that go to make up the posterior roots divide as soon as they enter the cord into two branches, a long ascending and a short descending fibre. Both of these fibres pass longitudinally in the posterior columns, the descending ones a short distance only before bending at a right angle to enter the posterior gray substance of the cord where each one terminates in an arborization. The ascending fibres are short and long. The former, although going in an opposite direction to the descending fibres, have a similar termination in the approximate gray matter. The long ascending fibres, on the other hand, traverse the entire length of the posterior columns and terminate by arborization around the cells of the nuclei of these columns, the columns of Goll and Burdach. The ascending and descending fibres give off lateral collaterals, which pass from the axis cylinder at right angles into the gray substance of the cord. The cells of the cord around which the collaterals break up and the arborizations take place are the columns of cells of the posterior and middle horns, the anterior horn cells, the cells of Clarke's columns, and the column cells of the opposite side. This makes up the peripheral (primary, first) sensory neuron.

From the axis-cylinder prolongations of the ganglion cells around which the peripheral sensory neuron terminates, there arise the conducting fibres of the central sensory neuron which go towards the cortex. Whether these axis-cylinder prolongations go the entire way to the cortex or whether they terminate in infracortical intermediate stations, from which their functional conductivity is transferred to a conducting fibre of a third order, is still an open question. It seems at least to be definitely settled that the pathway for tactile and muscular sense passes directly to the cortex of the central convolutions. Flechsig has demonstrated this in a most exquisite way by the embryological method. This pathway is the first central pathway of the projection system to be developed. At the eighth month of fœtal life there is seen a mass of medullated fibres occupying that portion of the internal capsule, injury of which in later life produces hemianæsthesia of the opposite side. This bundle passes from the nuclei of the posterior columns to the thalamus and lenticular nucleus on one side, through the posterior portion of the internal capsule to the central convolutions; on the other side the course lies through the cerebellum.

The secondary sensory neuron, forming the nucleocortical tract, extends in different pathways from nuclei in the cord and oblongata, through intermediate stations perhaps, to the cortex. They form

the fillet in its various divisions. The fibres of the central sensory neurons give off innumerable collaterals, which probably terminate, in great part at least, around cortical motor cells. What anatomical relationship exists between the cortical motor and sensory cells it is impossible to say at present. Theoretically many psychophysical phenomena would seem easy of explanation if some such arrangement as that which exists in the sensory and motor structures in the spinal cord, which mediates reflex action, could be posited—as, for instance, the passing of a fibre from a sensory axis-cylinder prolongation towards a cortical motor ganglion cell and arborizing around it.

The pathways of the nervous system for the conduction of impulses are divided into two great systems: the system of projection fibres and that of ssociation fibres.

The projection fibres are those which connect the cortex of the brain with the periphery and are classified into the projection fibres of the cerebrum and those of the cerebellum.

The association fibres are those which connect the various cortical areas one with another. They are of two kinds: the commissural fibres which connect similar areas on the two sides of the brain, and association fibres which connect asymmetrical cortical areas of the two hemispheres or parts of one region of the cortex with parts of remote regions of the same hemisphere. The projection fibres of the cerebrum form the so-called pyramidal tract or motor pathway. It begins in the large pyramidal cells of the ascending parietal convolutions, those going to the lower extremities being in the upper third of these convolutions, those for the upper extremities in the middle third. The fibres pass into the white substance, the centrum semiovale, forming a part of the corona radiata which passes into the posterior limb of the internal capsule, thence through the part of the cerebral peduncles called the crusta or pes pedunculi into the pons where they form well-defined pyramidal tracts. They continue through the pons, gradually approximating each other in the oblongata, where they form very distinct bundles on the ventral surface. At the distal end of the oblongata, the decussation of the pyramidal tracts begins, by which the columns of one side cross over to the other. This decussation is 6 mm. long and is complete at about the level of the second cervical nerve. The decussation is not entirely total. A very small number of the fibres continue their course along the anterior longitudinal fissure down the central surface of the cord as far as the lower dorsal region. These constitute the anterior pyramidal tracts. The decussated projection fibres take up their position laterally on the other side of the spinal cord, immediately in front of the entering posterior roots and separated from the periphery in the

upper cord by the direct cerebellar tracts. The crossed pyramidal tract gradually becomes smaller as it passes down the cord, on account of the fact that it is continually terminating by sending collaterals into the anterior horns which break up around the motor cells there situated, and eventually by the terminal arborization of the nerve fibre itself. The remainder of the pyramidal tract is constituted by the peripheral motor neuron, the ganglion cell of the anterior horn; its axis-cylinder prolongation, collaterals, and end organs.

The motor tract for the cranial nerves cannot be considered here, except to say that, according to some very recent investigations (Schlesinger, Bechterew), the tract from the fillet to the crusta ("Bündel von der Schleife zum Fusse") is probably the central motor tract for the cranial nerves.

The nucleocortical sensory tracts for the transmission of the different forms of sensory stimuli are not well defined. As we have already said, the one of which we are absolutely certain is that of muscle sense. The nuclei of the posterior columns in the oblongata are unquestionably the terminal stations of the peripheral sensory neurons which convey this form of sensation. From here the pathway is the interolivary fillet, the principal fillet. The pathway for tactile sense may travel with this, but it has not been so clearly elucidated.

The pathway for the higher senses is more definitely known. The central sensory conducting pathway or fillet, after it leaves the oblongata, divides into three parts: the chief or mesal portion going through the crusta to the cerebrum, the upper fillet to the superior quadrigeminal body and occipital region of the cerebral hemisphere, the lower fillet to the inferior quadrigeminal body of the same side.

The commissural fibres of the brain unite symmetrical portions of the two hemispheres and make up the corpus callosum and the anterior commissure. Until recently it was taught that the corpus callosum contained only such fibres, but Ramon has shown that it contains association fibres as well. The association fibres are divided into the long and the short association fibres, the long fibres uniting distant parts of the same hemisphere, the short fibres (fibræ propriæ) doing a similar service for adjacent parts. The long association fibres are divided into the superior longitudinal fascicle (fasciculus longitudinalis superior or arcuatus), which unites the frontal with the occipito-temporal, chiefly with the temporal lobes; the subcallosal longitudinal fascicle, which reinforces the connection just mentioned and connects the frontal with the occipital lobe; the inferior longitudinal fascicle, which unites the temporal and occipital lobes; the uncinate fascicle, which connects the inferior frontal with the tem-

poral lobes; the cingulum, which connects the anterior portion of the olfactory bulb with the frontal lobes and the hippocampal gyrus with the temporal lobes; and the vertical fascicle (perpendicular occipital bundle of Wernicke), which unites the inferior temporal lobes with the posterior part of the fourth temporal convolution, that formerly described as the first occipitotemporal lobe and sometimes as the fusiform gyrus.

Arterial Blood Supply of the Brain.

In this consideration of the arterial supply of the brain, in its relation to pathological conditions, the arteries that enter into the formation of the circle of Willis will be considered first; secondly, the arteries that branch from these, entering the basal portions of the brain; thirdly, the cortical branches; fourthly, the arterial supply of the cerebellum and oblongata, and finally certain pathological conditions that are determined in part by the mode of distribution of these arteries in relation to arterial volume and pressure.

The internal carotid artery, after giving off the ophthalmic branch, is connected, where it passes at the base of the brain near the inter-peduncular space, by means of the posterior communicating artery with the posterior cerebral artery of its own side. The anterior cerebral artery is given off just in front of this and passes forwards and inwards above the optic nerve, in front of the optic chiasm, to the beginning of the longitudinal fissure between the cerebral hemispheres. As it enters the fissure, the anterior communicating artery unites it with its fellow of the other side. The two posterior cerebral arteries are derived from a common stock, the basilar artery, which itself is formed by the union of the right and left vertebral arteries which are given off from the upper part of the subclavian. Thus the circle of Willis is a communicating channel closed posteriorly by the basilar artery, laterally by the two posterior communicating arteries, the internal carotids and the two anterior cerebral arteries, and anteriorly by the anterior communicating artery.

The supply of the basal parts of the brain is given by the following arteries: 1. The anterior choroid artery, a branch of the internal carotid, which runs outwards and backwards in the cleft between the temporal lobe of the cerebral hemisphere and the crus cerebri to reach the choroid plexus in the descending horn of the lateral ventricle. According to Kolisco, the anterior choroid artery supplies the posterior arm of the internal capsule with the lamina medullaris of the thalamus, the white substance back of the internal capsule as far as the roof of the descending horn, the inner

lenticular segment, the uncus, the posterior half of the optic tract, the lateral choroid plexus, the ependyma of the posterior and lower parts of the lateral ventricle, the greater part of the tail of the caudate nucleus, and exceptionally the external parts of the upper half of the thalamus. The branches in the plexus and tela anastomose freely with others near by, but those to the brain substance are terminal. This condition favors hemorrhages and softening of the brain substance. But the posterior arm of the interior capsule is partly supplied by the middle cerebral and posterior communicating artery. 2. The anterior mesal group of central arteries, branches of the anterior cerebral artery which pierce the anterior part of the anterior perforated space and supply the anterior extremity of the caudate nucleus, together with two or three unimportant twigs which are commonly included in this group, passing from the anterior communicating artery to the lamina cinerea, the rostrum of the corpus callosum and adjacent parts of the septum lucidum. 3. The anterolateral arteries which are given off from the middle cerebral arteries immediately after leaving the carotid artery, passing through the foramina of the anterior perforated space to the base of the corpus striatum, forming the most important supply of that region. The division of these arteries commonly follows that of Duret into the lenticular arteries, supplying the inner and middle segments of the lenticular nucleus and internal capsule; the lenticulostriate arteries, supplying the outer segment of the lenticular nucleus and external capsule, and the caudate nucleus; and the lenticulooptic arteries, supplying the outer and posterior parts of the lenticular nucleus and the outer parts of the optic thalamus. One of the lenticulostriate arteries, larger than the rest, and ending in the caudate nucleus, has been called by Charcot the artery of cerebral hemorrhage, because of its being so frequently a seat of cerebral hemorrhage. 4. The posteromesal central arteries and the posterolateral central arteries, both offshoots from the posterior cerebral artery, the former ascending through the posterior perforated space to the inner part of the crus cerebri, the optic thalamus, and wall of the third ventricle, the latter arising on the outer side of the crus and supplying the corpora quadrigemina and the hinder part of the optic thalamus, giving off two or three small twigs to the crus as it passes over that body. 5. The posterior choroid arteries, composed of one or two branches to the velum interpositum and upper part of the choroid plexus.

The superficies of the cerebral hemispheres is supplied by the branches of three main cerebral arteries. The four branches of the anterior cerebral artery comprise (1) two to four small inferior internal frontal arteries to the internal orbital convolutions; (2)

the anterior internal frontal artery, supplying the lower part of the marginal convolutions, and the superior and middle frontal convolutions; (3) the middle internal frontal arteries, distributed to the callosal convolution and upper part of the marginal convolution; (4) the posterior internal frontal arteries supplying the quadrate lobule, and giving off the artery of the corpus callosum running backwards on the upper surface of that body to its posterior extremity. The cortical branches of the middle cerebral artery or Sylvian artery, the continuation of the main trunk of the internal carotid, are (1) the inferior external frontal, distributed to the outer part of the orbital surface of the hemisphere and adjacent frontal convolutions; (2) the ascending frontal to the convolution of the same name and to the root of the middle frontal convolution; (3) the ascending parietal to the parietal convolution and the fore part of the superior parietal lobule; (4) the parietal temporal which runs backwards in the posterior limb of the fissure of Sylvius and ramifies upwards over the inferior parietal and the hinder part of the superior parietal lobule, and downwards over the superior and upper part of the middle temporal convolution. The posterior cerebral artery gives off three cortical branches: (1) The anterior temporal, distributed to the anterior part of the uncinate gyrus and its immediate vicinity; (2) the posterior temporal to the middle part of the uncinate, to the external occipitotemporal and to the lower temporal convolution; and (3) the occipital, lying in the calcarine fissure and supplying the occipital lobe on its inner and outer surfaces.

The supply of the cerebellum is given in part by two branches from the basilar artery: the anterior inferior cerebellar arteries which pass backwards, one on each side, to the anterior part of the under surface of the cerebellum, anastomosing with the inferior cerebellar branches of the vertebral arteries; and the superior cerebellar arteries, which reach the upper surface of the cerebellum, and there divide into branches some of which extend outwards and backwards along the superior vermis to reach the circumference of the cerebellum, where they are supposed to anastomose with branches of the inferior cerebellar artery. Others pass inwards to supply the vermis, the valve of Vieussens, and in part the velum interpositum. Another artery supplying the cerebellum is the (posterior) inferior cerebellar artery, the largest branch of the vertebral artery. It divides at the fore part of the vallecula into two branches, which divide again and are distributed to the oblongata and choroid plexus of the fourth ventricle, in part passing over the surface of the cerebellar hemisphere to the inferior vermis, anastomosing with the terminals of the opposite artery and with those of the superior cerebellar artery. The bulbar branches

of the vertebral artery alone remain requiring special mention here. They comprise numerous small branches supplying the oblongata, penetrating mainly along the roots of the nerves and in the anterior median fissure. The important investigations of Adamkiewicz have laid bare the minute details of the arterial distribution of this region. They will be referred to in the section on diseases of the oblongata.

The results of a few important investigations as bearing upon conditions of distribution that may modify pathological developments in the cerebrum will now be mentioned. According to Löwenfeld, the left carotid is larger than the right, and, according to Quain, the left vertebral is generally larger than the right. Mendel has found that pressure in the cortical vessels is materially less than in the carotid, from which they spring, while in the striatial or lenticular arteries it is not materially less, perhaps because of the terminal character o these vessels as well as of their less circuitous distribution. Kolisco finds that the central branches of the anterior cerebral arteries are of two sorts. They are either short and of small calibre, delicate in texture and thus favoring the development of thrombosis, such as supply the anterior portion of the globus pallidus and of the anterior arm, of the internal capsule, and also the genu; or they are long branches without ramifications, such as pass through the anterior perforated space into the head of the nucleus caudatus, the anterior arm, and the basal portion of the anterior part of the putamen. This latter mode of distribution favors softening and makes hemorrhages difficult. The territory supplied by the anterior carotid artery, as has been shown above, is richly furnished with blood from other arteries, in consequence of which softenings in that region are mostly partial and limited to the middle third of the posterior arm of the internal capsule. It hardly needs to be pointed out that focal lesions due to hemorrhage or softening are more frequently found in the striatial region than in the territory of cortical distribution, or within the choroid plexuses of the ventricles, because of the absence of anastomosis and because of the very direct connection of the lenticulostriate arteries with the main trunk of the internal carotid.

The relation of intracranial pressure to arterial pressure has been the subject of an extensive investigation by Grashey. Contradicting the view of Adamkiewicz, Grashey's results have demonstrated that compensation for augmented intracranial pressure is not found in compression of brain substance, but in that of the termini of the veins, in the sinuses, and in the expansion of the capillaries. The compression of the veins induces a venous pulsation and is the primary cause of the venous pulse that is sometimes felt in the internal jugular vein. Increase in aortic pressure induces, with elastic cere-

bral vessels, a marked increase in intracranial pressure and a smaller increase in the pressure upon the walls of the vessel, while under similar conditions the opposite effect is produced with inelastic vessels. Consequently hemorrhage occurs most frequently with inelastic vessels. Pathological brain pressure begins to exist with a pulsation of cerebral veins. This need not take place unless a considerable increase in amount of cerebrospinal fluid occurs, according to results of Naunyn and Falkenheim, owing to a compensation for such augmented intracranial pressure, due to the resorption of the cerebrospinal fluid which takes place more rapidly than its secretion. Although thinning of the blood increases the secretion, yet an increase in the blood pressure in the cerebral arteries does not affect either secretion or resorption. Compensation may also be obtained by a carrying off of cerebrospinal fluid through the venous tract, direct passages having been found by Reiner and Schnitzler, who believe these to be in part through the Pacchionian granulations. Cavazzani finds evidence that the sympathetic produces both vasomotor constriction and dilatation of the cerebral artery.

CEREBRAL LOCALIZATION.

Historical Note.

The history of cerebral localization dates back to the beginning of the present century, but the subject began to be placed on a substantial foundation by Fritsch and Hitzig, who discovered the irritability of certain parts of the cortex of the brain to the galvanic current in 1870. Previous to that time, the attempt to associate definite parts of the brain with performance of certain functions had been successful only in one instance, that of motor speech centre discovered by Broca. Early in the beginning of the nineteenth century Franz Josef Gall, whose name is associated now almost exclusively with phrenology, taught that the cerebral convolutions were the seat of psychical activity, and that the individual convolutions of the brain were not of equal participation and importance in these functions—in other words, that specific mental faculties could be localized in certain convolutions. This was an immeasurable advance on the teachings of Descartesis, Sömmering, and other predecessors and made an approximation to the localization teaching of the present day.

The physiologist Flourens (1842) was led from experiments on animals to teach that the cerebral hemispheres were the seat of perception and of the will, and that these faculties were evenly distributed, not localized as taught at present. This contention was denied by

Gall and his pupil Bouillaud, by Marc Dax and especially by Broca, who in 1861 localized the speech area in the foot of the third left frontal convolution.

From 1863 to 1870 no epoch-making discovery was made in cerebral localization, although the clinico-pathological work of this period, in particular that of Hughlings Jackson (1861), was preparatory to the interpretation of the results of experiments by Fritsch and Hitzig (1871), Ferrier (1873), Munk, and their disciples.

The mere enumeration of those who have materially contributed to our present knowledge of cerebral localization would require a great amount of space. It is fitting, however, that a few names indissolubly associated with the subject should be mentioned; such are Ferrier, Horsley, Beevor, Schäfer, and Gowers in England; Hitzig, Munk, Flechsig, Kussmaul, Lichtheim, Wernicke, Nothnagel in Germany; Luciani, Albertoni, Seppilli, and Tamburini in Italy; Broca, Charcot, Franck and Pitres, and Déjerine, in France; Seguin, Dana, and Mills in America; Henschen in Denmark; Bechterew in Russia, and v. Monakow in Switzerland. These names and others must be frequently mentioned even in an article which makes no pretension to exhaustiveness.

The relative importance of the methods contributing to the present status of cerebral localization varies according to the one making the estimate. Some deny the really great service of experimental physiology, while others give it undue praise. It must be admitted that experimental physiology has given valuable service, first by directing the clinician's attention directly to the subject and by stimulating his interest in the anatomico-pathological study of localized brain disease; and second, by corroborating inferences drawn from the study of his cases and particularly by homologizing them with conclusions reached from experiments on the lower animals. Nevertheless, the service which experimental physiology has rendered does not compare with that which has accrued from the careful, intelligent observation of clinical phenomena and their interpretation in the light of post-mortem findings.

The literature of the subject is colossal. In the preparation of this article use has been made of the numerous *résumés* up to 1890. The contributions to the subject since that date and many previous to it have been studied in the original and carefully analyzed.

Localization in the Cortex of the Brain.

The cortex of the brain may be divided, according to its determined physiological functions, into areas for motor representation, for special sense representation, and for the representation of sensory per-

ception. When one or more of these areas are diseased or encroached upon, the result is a disturbance of function, either an exaggeration or an inhibition, manifested in disorder or destruction of motion, of the special senses, or of general sensory interpretation. It matters but little what the nature of the lesion is—the location of the lesion is all important.

Our knowledge of the motor areas of the brain, especially the areas for the extremities and the face, is more settled than that of any of the other areas. Of the special senses the visual areas have been most reliably established, although cortical representation for hearing is nearly unassailable. Of the special senses the one most unsatisfactorily made out is the sense of touch. Much confusion has resulted from reports of observation and experiments purporting to contribute to the localization of the tactile sense, in which no conception seems to be had of the difference between tactile sense and other forms of sensation mediated by the skin. And they are not infrequently all spoken of in a general way as "common sensation" or "disturbance of common sensation."

The localized areas of the brain are: (1) for motion; (2) the special senses: (*a*) sight, (*b*) hearing (audition), (*c*) taste, (*d*) smell, and (*e*) touch; (3) for sensations including muscular sense, sense of position, thermal sense, sense of pain, of pleasure, of hunger, of desire, etc., as well as static and kinæsthetic senses; (4) for complex psychical processes.

The motor area of the brain is located, on the external surface of each hemisphere, in the convolutions lying immediately in front and behind the sulcus centralis or fissure of Rolando, the ascending frontal and ascending parietal, or pre- and postcentral convolutions; while on the mesal surface of the brain it is situated anterior to the upper end of the fissure of Rolando in the paracentral lobule. To this motor area may probably be added the marginal part of the first frontal on the mesal surface and the posterior portions of the first and second frontal on the lateral surfaces (Fig. 9). The motor area on the external surface of each hemisphere is subdivided into an area for representation of the lower extremity, one for the upper, and one for the cephalic extremity. These are situated, in the order mentioned, from above downwards, the leg area occupying a greater amount of cortical surface antero-posteriorly than either of the others, but a considerably narrower surface from above downwards. The anatomical limits of each of these subdivisions cannot be defined with mathematical accuracy, but in a general way it may be said that the limitation below between the leg and the arm area is the first sulcus, from above downwards in the ascending frontal and ascending parietal convolu-

tions the superfrontal and the subfrontal fissures. The arm area is situated below this, it has narrower cortical confines antero-posteriorly than the leg area and has greater representation in the as-

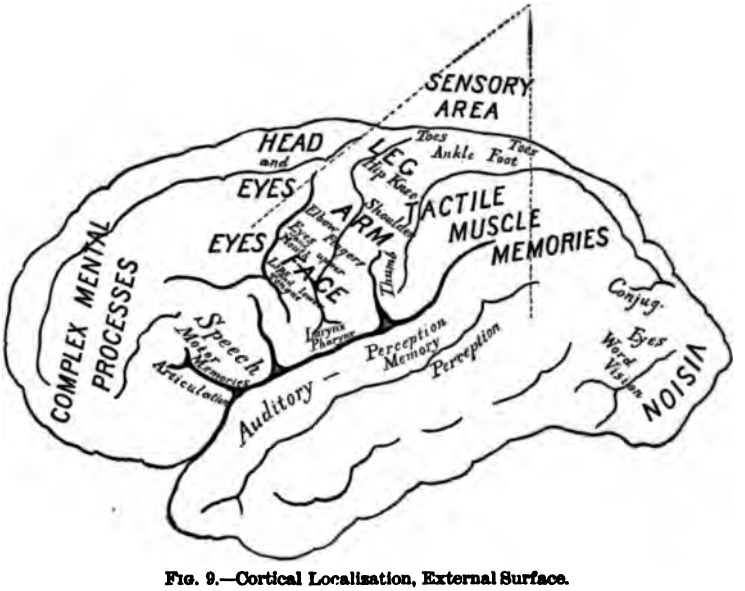

FIG. 9.—Cortical Localization, External Surface.

cending parietal convolutions. The area for the cephalic extremity is situated just above the beginning of the posterior limb of the fissure of Sylvius, between this and the lower border of the arm area.

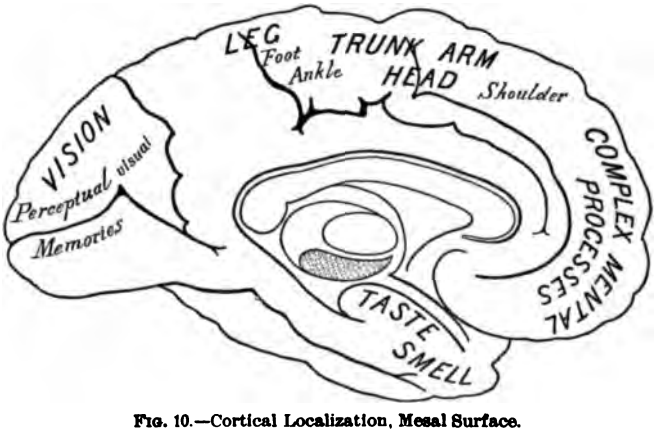

FIG. 10.—Cortical Localization, Mesal Surface.

The trunk has not very much representation on this surface of the brain, and what it has is not well differentiated.

On the mesal surface of each hemisphere the motor areas which

have been carefully outlined and defined are those for the arm, trunk, and leg. On this surface the cephalic extremity has but small representation. The arm area on this surface is situated anteriorly, the leg area posteriorly, and the trunk area between. The motor area is limited on the mesal surface of the hemisphere almost entirely to the paracentral lobule (Fig. 10). There is an area on this surface, however (not shown in the accompanying cut), irritation of which will cause twitching of muscles of the head; it is situated in front of the arm area in the marginal frontal convolution.

Each of the three great subdivisions of the cortical motor area on the external surface of the brain has been subdivided into areas for the movement of individual and component parts; such as movement of the thumb in the arm area, movements of the toes in the leg area, movements of the eyelids, of the jaw, of the components of the larynx, etc., in the head area. The arm area has been differentiated into an area for the shoulder, for the elbow, the wrist, and the thumb. These are situated from above downwards in the order mentioned: that for the shoulder occupying the highest part of the arm area, that for the thumb the lowest, and bordering on the face area, the wrist area being just above the thumb area, and the elbow area between this and the motor cortical representation of the shoulder. Destruction of the arm area causes brachial monoplegia. Taylor records a case in which traumatic ablation of this area produced complete brachial monoplegia which after nearly two years almost completely recovered, the recovery proceeding from the shoulder towards the fingers, the latter never completely regaining their power.

The subdivisions of the arm area as well as those of the remainder of the motor area about to be considered have been determined by electrically stimulating the cortex of the brain in man and in apes and carefully noting the muscular responses, and by the study of localized lesion of these areas, before and after death. If a bipolar electrode carrying a weak faradic current be placed on the pia just in front of the fissure of Rolando at the level of the junction of the upper and middle thirds of the central convolutions, a contraction of the thumb will follow. If it is placed a little above and posteriorly, contraction of the fingers and wrist will result, while if placed higher up, that is towards the longitudinal fissure, contraction of the elbow and finally of the shoulder will occur. On the mesal surface of the hemisphere the topographical representation of the arm is, as has previously been stated, limited to a comparatively small space situated mostly in the anterior end of the paracentral lobule and the adjacent marginal frontal convolution.

The motor area for the leg is subdivided into an area for the hip and thigh and an area for movements for the leg and toes with some differentiation for the toes, especially the large toe. The surface area of representation of the lower extremity is somewhat greater on the mesal surface of each hemisphere than on the lateral. Faradic irritation of the different portions of the leg area as well as observation of localized lesions tend to show that the area for movements of the thigh is situated towards the anterior end of the leg area on the lateral and mesal surfaces, while that for the knee and foot is situated towards the posterior end of the same areas. If the leg area on the lateral and mesal surfaces of the hemisphere be divided into thirds, the junction of the middle and posterior thirds, according to Beevor and Horsley, indicates the seat of representation of the large toe, while that for the small toes is not limited to such narrow confines; both the anterior and the posterior ends of the leg area seem to have individual representation for the latter. Lesion of the mesal surface localized and confined to that surface is of the greatest rarity, but faradic excitation of the posterior part of this area seems to cause contraction of the flexors of the toes and ankles.

Numerous striking clinical examples of the localization of lesion in the leg area have been put on record, especially by Seguin, by Hun, by Koenig, and by many others. In a case reported by Hun, epileptiform manifestations following trauma were signaled by spasm of the left foot. Later there was left hemiparesis. The autopsy showed a hard, ovoid tumor 37 by 30 mm. in the upper extremity of the postcentral gyrus near the longitudinal fissure. Seguin's case was almost exactly similar to this. In a case recorded by Mills, a small gumma involving the leg area both in front and behind the upper end of the fissure of Rolando caused severe attacks of left-sided spasms, beginning with twitchings in the opposite foot and toes. Horsley has also published the history of several cases in which the differentiation of the leg area has been strikingly shown.

The seat of representation for the trunk muscles is not very well known. What is known of it has been made out principally by experimentation, it being extremely uncommon to encounter clinical and pathological evidence bearing on this point.

As has already been said, the motor area for the muscles of the trunk is more extensive topographically and more distinctly represented on the mesal than on the external surface of the hemispheres. In the former it occupies a relatively large space in the paracentral lobule between the areas devoted to the representation of the arm anteriorly and of the leg posteriorly. On the lateral surface of the hemispheres there would seem to be a small representation for the

trunk, principally in the ascending frontal convolution just anterior to the leg area. There are cases recorded which tend to show that the representation for the trunk on the mesal surface is largely for the abdominal muscles and that on the lateral surface for the chest and back muscles.

The only differentiation of the trunk which has as yet been attempted is that for the movements of the vagina. This has been located by Bechterew and Mislawski in the anterior margin of the motor region, presumably the trunk fields, in rabbits, and in the sigmoid gyrus, a name given to the convolutions about the cruciate sulcus, especially in animals, such as dogs. The movements which faradic irritation of these areas causes are those of contraction and relaxation. The cortical centre for the sphincter ani is stated by Bechterew to be somewhat back of the nucleus cruciatus, in the posterior part of the sigmoid convolution near its external margin.

The cortical area for the cephalic extremity has taken longer to arrive at satisfactory differentiation than any of the others, but the observations and experiments that have been published in the past few years show that labor in this direction is beginning to be productive. The area of cortical motor representation for the cephalic extremity is subdivided into that (1) for movement of the head; (2) movement of the eyes; (3) movement of the pupils; (4) movement of the face; (5) laryngeal movement; (6) movement of the lower jaw; (7) movement of chewing and swallowing, and lingual movement; (8) the muscles of speech which are represented in this area. The area for the cephalic extremity admits of two great subdivisions, the head area and the face area. The latter is by far the better defined and represented. The former, the head area, is situated on the lateral surface in the cortex of the posterior part of the first and second frontal convolutions, those parts adjacent to the upper end of the ascending frontal convolution and in the corresponding part of these same convolutions, especially the first frontal on the mesal side.

The face area is at the lower two-fifths of the central convolutions on the lateral surface. The anatomical limitations put upon the face area by different observers vary considerably. Brissaud has recorded a case in which there was right-sided facial paralysis with muscular atrophy, hyperæsthesia, and slight ptosis and dilatation of the pupil subsequent to a right-sided hemiplegia and aphasia which had nearly disappeared. The brain showed a focus of softening in the lower part of the left ascending parietal convolution just behind the inferior extremity of the fissure of Rolando. In the extreme lower end of the ascending parietal convolution experimentation seems to show that there is a motor centre for the platysma myoides muscle and clinical

evidence confirmatory of this is not lacking. Bramwell has published a case in which spasm always began and was practically confined to the right platysma, in which a spicule of bone was found irritating the inferior margin of the postcentral convolution. Representation of the face on the mesal surface, if it has any, is unknown. The localization of the face area is based largely on pathological and clinical evidence, and is therefore more trustworthy than that of almost any other area. It is subdivided into an upper and a lower portion. In its upper and anterior portion are the centres for the movement of the orbicularis and the frontalis muscle and the angle of the mouth, while in the lower portion are probably differentiated the centres for the lips, tongue, throat, and larynx, the area for the latter being situated on a plane posterior to that for the lips and tongue. It may be stated as proven that facial and lingual monospasm or monoplegia indicate a very limited and well-defined lesion of the inferior and of the so-called motor region on either side (for the representation is bilateral) and particularly of the ascending frontal. This area is considered the cortical origin of the hypoglossal nerve. Bernheim has contributed some very satisfactory evidence to the localization of lingual movements. In a case in which there was well-marked deviation of the tongue to the right, no other paralysis and no aphasia, there was found after death a sarcomatous blood cyst, 5–6 mm. long, located towards the inferior border of the foot of the left precentral gyrus, about 6 mm. behind the sulcus which separates it from the third frontal.

The cortical area of the throat and laryngeal muscles has been carefully studied by Krause, Masini, Horsley, and Semon, and their studies have corroborated clinico-pathological claims. In the ape the centre for the larynx is situated in the anterior part of the foot of the ascending frontal convolution. In man this centre is situated in the frontal operculum—that is, in the region between the anterior ascending and the anterior horizontal rami of the fissure of Sylvius. Laryngeal representation is bilateral, but the preponderant representation for the vocal cords of one side is in the opposite hemisphere; still it is not denied that this hemisphere has some influence over the vocal cords of the same side. The evidence that has been produced to show the predominant influence which one hemisphere exercises on the movements of the contralateral cords is of the most convincing nature. Most of the German authors incline to the teachings of Semon and Horsley, that each hemisphere governs the movements of both vocal cords, but the testimony given by the cases of Brissaud and of Wallenberg seems to me conclusive in favor of the view above stated. In the case of the last-mentioned author, in which there was laryngeal paralysis of the left side, the projection fibres

from the portion of the cortex in which we place the laryngeal centre in man were entirely destroyed. Some clinico-pathological observations tend to show that the adjacent third frontal convolution is also the seat of laryngeal representation to a slight degree. Seguin's case, in which in addition to slight left hemiplegia and motor aphasia there was inability to use the muscles of the pharynx and larynx, showed on autopsy lesion of the third frontal convolution, on both sides, and slight change in the foot of the adjacent ascending frontal. Déjerine has published the result of two autopsies which point with great positiveness to the fact that the cortical laryngeal centre occupies the inferior end of the ascending frontal convolution, and Garel has published an observation which is strikingly corroborative of this contention. The important clinical feature was paralysis of the vocal cords. Autopsy showed localized meningoencephalitis of the lower end of the right ascending frontal gyrus, and at the foot of the third frontal gyrus were two small spots of softening.

Attempts have been made to localize centres for the muscles of mastication and deglutition. Langer, and likewise Lepine published cases which they believed went to show that such areas were in the island of Reil. The experiments of Bechterew, of Ostankow, and of Rethi deny this. The experiments of the last-named observer made on dogs show that excitation of an area situated at the anterior end of the second fissure at a point where the latter would meet with an imaginary prolongation of the sulcus cruciatus downwards and forwards caused movements of swallowing. Excitation of a spot just above medially of this swallowing centre, therefore the anterior end of the second frontal gyrus, causes accelerated breathing with prolonged expiration. The proximity of these two centres is in accordance with the physiology of the act of swallowing. Both centres are represented in each hemisphere. Rethi believes that he has seen a confirmation of his claims in man. The patient suffered from attacks of spasm of the gullet. Trepanation exposed the lower half of the second frontal convolution. Faradic excitation of the brain in the most anterior part of the wound produced spasm of the gullet.

The centres for the movements of the lower jaw have been determined experimentally to be situated in very close connection with those for laryngeal, pharyngeal, and lingual movements in the ascending frontal convolution about at the junction of the upper and middle thirds of the face area. These claims have not yet received clinical or pathological confirmation.

The centre for the action of the levator palpebræ superioris, which causes elevation of the upper eyelids, has been isolated by the experi-

mental research of Ferrier, of Beevor and Horsley, and of Mott; but the location of the centre which they claim does not coincide with that posited by clinico-pathological evidence, or, at least, does not seem to coincide. Irritation experiments seem to prove the existence of a centre for elevation of the upper eyelid in the posterior portion of the second frontal convolution contiguous to the centres for movement of the head and eyes, situated there and in the adjacent convolutions. The histories of a number of cases having inability to raise the upper eyelid have been recorded, in which lesions have been found occupying the angular gyrus or its immediate environments. The interpretation put upon these cases, and the one that most satisfactorily explains the apparent discrepancy between them and the results of experimentation, is that the irritation by the lesion in this region causes a reflex contraction of the levator palpebræ superioris through sensory stimulation. Beevor and Horsley have shown, in the brain of the monkey, that there is a large area for the representation of the head and eyes, which reaches from the longitudinal limb of the fissure of Sylvius below into the frontal convolutions. It is probable that this area represents the motor centres for movement of these structures in man. The experimental as well as the clinical evidence is not at all conclusive or convincing. Ferrier found that irritation of the superior and middle frontal convolutions caused lateral movements of the head and eyes to the opposite side, with dilatation of the pupils, and that a similar movement resulted from irritation of the angular gyrus and the superior temporal gyrus; and he explains this by saying that the latter are attention movements, and comparable to those obtained by stimulating the motor areas. Risien Russell found special centres, not only for lateral but for upward, downward, upward and outward, downward and inward, and convergent movements of the eyeballs in the cerebral cortex. Ablation of these centres, after having been determined by excitation, was followed by paralytic symptoms, which, however, after some time disappeared, but which, it was remarked, would always recur during narcosis.

Cerebellar centres controlling the eye movement have also been found, and the effects of the cerebral cortex upon ocular movement seem to be antagonistic to those of the cortex of the cerebellum; that is, if the cerebellar lesion paralyzes the movement in one direction, lesion of the cerebral cortex will paralyze it in the opposite direction, the result being that the position of the eyeball is unchanged.

Mott believes that the cortical area of the frontal lobe in monkeys, which when stimulated gives rise to associated eye movements, can be divided into three zones: 1. A middle zone immediately below the horizontal part of the precentral sulcus, irritation of which is followed

by simple lateral deviation to the opposite side; 2. A zone immediately above this, which may extend to and include part of the marginal gyrus, irritation of which gives downward movement of the eyes, combined with lateral deviation; 3. A motor zone immediately below (1), irritation of which gives upward inclination. He also believes that bilateral faradization of identical spots in the visual area produces results similar to those obtained in the frontal area, but that weak stimulation of the frontal area readily overcomes strong stimulation of the occipital area.

There is considerable clinical evidence against the localization of a cortical motor centre for movements of the eyeballs in the second frontal gyrus, one of the most important cases being by Coats, in which a fracture in the left temporofrontal region allowed cerebral hernia to form. The symptoms were complete and permanent paralysis, with contracture of the right arm; temporary paralysis of the right side of the face and lingual deviation to the right; paresis of the right leg and some motor aphasia; later, convulsive attacks, beginning in the right cheek and arm. The lesion was evidently injury of the precentral gyrus, and probable tearing of the second and third frontal convolutions. This case has been given considerable importance by Seguin, as negatory of Ferrier and Horsley's claims; but I cannot agree with him, because the ocular deviation may have been present early, and later the automatic nuclear centres for the eye muscles caused its rectification and disappearance. Mills has published an observation showing that application of the faradic current to the posterior extremity of the second frontal convolution caused distinct deviation of the head to the opposite side.

In summarizing briefly the present status of motor localization, I shall first state the conclusions of Beevor and Horsley, which are based on experimentation and which have been recognized by Ferrier, and then repeat the most important conclusions of clinico-pathological evidence.

Beevor and Horsley's conclusions are:

1. The centres for the lower extremity are arranged progressively from before backwards, the most anterior centre for the highest segment of the extremity and the centres for the rest of the extremity in serial order, as if the lower extremity lay lengthwise on the convexity of the hemispheres.

2. The centres for the upper extremity are arranged in a similar way, but lengthwise along the fissure of Rolando.

3. The centres for movement of the head area are situated in front of the motor zone, properly speaking, in the foot of the first frontal convolution.

4. The centres for movement of the trunk, especially represented on the internal surface of the hemisphere, are between those of the thigh and shoulder.

5. The centres of the face are arranged progressively from above downwards, respectively, for the representation of the eyelids and the mouth in the inferior Rolandic region, as far as the lower extremity, at the level of the ascending parietal convolution.

6. The centre for the movements of the eyes is in front of the ascending frontal convolution on the same level.

7. The centres for the larynx, the pharynx, and for mastication are in the inferior extremity of the ascending frontal convolution.

Symptoms of localizing significance are spasms or twitchings, and paralysis of extremities or groups of muscles having a common function, or individual muscles on the side of the body opposite to the lesion. In a general way, it may be said that irritative lesions produce the former, destructive lesions the latter. Total hemiplegia of cortical origin is caused by destructive lesion of the ascending frontal and ascending parietal convolutions.

Partial destruction of these areas causes monoplegias. It is unnecessary to repeat that irritation of the motor region of one side produces hemispasm, and irritation of certain limited areas produces monospasm.

Brachio-facial monoplegia or monospasm is caused by lesion of the inferior part of the motor region; brachio-crural monoplegia or monospasm by lesion of the superior part of the same region; facial and lingual monoplegias and monospasms are caused, the latter by lesion of the foot of the ascending frontal convolution, the former by lesion of the cortex just behind the inferior extremity of the pre-Rolandic fissure. Crural monoplegia or monospasm alone is probably the result of lesion of the paracentral lobule. The annectant convolutions behind the pre-Rolandic fissure—*i.e.*, the anterior ascending limb of the fissure of Sylvius, together with the adjoining parts of the anterior, and to a much less extent the posterior, central convolutions—are the motor centres for the opposite half of the face and tongue, and also for the action of the muscles of deglutition and mastication. Destruction of the frontal operculum, the region between the anterior ascending and the anterior horizontal ramus of the fissure of Sylvius, will produce paralysis of the contralateral vocal cord and slight defect of innervation of the cord of the same side. The phonation centre is situated in the lower extremity of the ascending frontal convolution, in front of and adjacent to the motor speech centre.

Deviation of the ocular muscles is produced by lesion of the second frontal convolution in front of the ascending frontal. A centre

for movements of the eyes and the upper lid is probably located in that part of the second parietal convolution situated between the fissure of Sylvius and the first temporal sulcus.

Cortical Sensory Localization.

The confines of the sensory cortical area are not by any means definitely settled. The claims of experimental physiology are not in entire accord with those of clinical medicine; in some instances they are directly contradictory of the latter, but in many one is corroborative of the other. Complete destruction or disease of a cortical sensory area causes complete loss of all sensibility of the respective area; that is called cortical anæsthesia. These anæsthesias are denominated, according to the physiological function of the area involved, cortical blindness, cortical deafness, cortical analgesia, etc. Partial destruction of sensory cortical centres causes different sensory deviations, varying according to the centre. Partial destruction of the visual sphere will cause limited defect in the visual field, but partial destruction of the auditory field may cause either diminution or complete loss of perception for certain tones; while partial destruction of the centre for smell, for taste, and for muscular sense has not yet been carefully observed. The superiority of clinico-pathological evidence in the determination of centres is shown nowhere so clearly as in visual localization. In fact, it must be said that if we had been guided by the physiologists we should still be locating the seat of vision in the angular gyrus, which was for a long time, and is yet, except that a part of the cuneus is now included, the belief of Ferrier. The clinico-pathological experience of Seguin, Hun, Mills, Keen and Thompson, Haab, Huguenin, Nothnagel, and the clinico-physiological investigations of Monakow, Henschen, Déjerine, and others, have settled the question of visual localization beyond peradventure.

Visual Localization.

Ferrier for a long time contended that the angular gyri were the visual areas, and that the occipital lobes were of comparatively slight importance in visual representation. Munk limited the area to the occipital lobe. Luciani extended it to the angular gyrus. The evidence produced by clinicians has refuted these teachings; the occipital region is now considered to be the principal or primary visual area, and that portion of the inferior parietal convolution known as the angular gyrus to be the area for secondary visual residua. Ferrier holds at present that although the visual centres cannot be localized in the

angular gyrus, they include the angular gyrus and the occipital lobes, a region which he calls the occipitoangular region. Experimentation on the posterior part of the brain in animals seems at least to have proven that destruction of the angular gyrus as completely as possible causes no perceptual defect of vision and no loss of movement of the eyeballs. Such were the conclusions of Schäfer and Brown. Destructive lesion of the occipital lobe always causes lesion in relatively similar parts of both eyes—that is, destruction of one cuneus causes lateral homonymous hemianopsia, and destruction of both cunei causes permanent blindness. These are practically the conclusions of Horsley and Schäfer.

In a general way, these conclusions are in accordance with clinical experience. The cortical seat of primary visual representation—that is, for retinal projection—is in the occipital lobes, and the representation is a bilateral one. The retinal projection in the visual area was for a long time stubbornly contended for by Munk, and its admission is becoming more universal since Henschen claims it on pathological grounds. The details of this retinal projection or expansion are in great measure unknown, although it is claimed by some, especially by Henschen and Wilbrand, that there is individual representation not only for halves of the retina, but for quadrants and sextants. The former narrows the visual centre to a more limited space along the calcarine fissure than any other investigator, and he is of the opinion that, as in a transverse section through the optic radiations the optic fibres of the dorsal homonymous retinal quadrants are dorsally, the ventral ventrally, and the muscular fibres mesally, so the cortex of the superior border of the calcarine fissure, the cuneus portion, is for the dorsal homonymous retinal quadrants; the inferior border, the lobus lingualis portion, represents ventral retinal quadrants, and the cortex at the bottom of the calcarine fissure is for the macula lutea.

Against the acceptation of a definitely arranged retinal projection on the visual sphere no conclusive evidence can be advanced; but the weightiest is that recently furnished by H. Sachs, from the microscopical study of Förster's case of bilateral hemianopsia with relative freedom of the macula lutea. In this case there was found double-sided destruction of the calcarine fissure, except in the posterior third, where it was tolerably well preserved. Yet in one of Henschen's cases on which the conclusions are founded, there was disease of this posterior part with intactness of the macula. It must be borne in mind, however, that the macula lutea, the fixation point, has separate representation, and that it is represented for each eye in both cunei. Monakow says there is no projection from the retina into the cere-

bral cortex, but that the centre is mobile, so that a new centre can form of itself if the peripheral cortical centre be destroyed; but it is impossible to hold such a claim in the face of such cases as have been reported by Hun, Seguin, Déjerine, Henschen, and Wilbrand, in which the hemianopsia was permanent.

It is very probable that the primary visual centre occupies only the medial side of the occipital lobe, and that its boundaries are, above, the upper margin of the hemisphere; below, the inferior border of the third occipital convolution; behind, it extends to the apex of the occipital lobe, and anteriorly to the parieto-occipital fissure. Thus it will be seen that this includes the divisions of the occipital lobe known as the cuneus, the lingual, and fusiform lobules. Vialet, who has studied this matter with care, believes that hemianopsia may result from lesion of any of these parts. His conclusions agree in the main with Seguin's, and the weight of clinical evidence seems to bear out the belief of the writer. Seguin, as well as Henschen and many clinico-pathologists, believes the visual centre to be limited to the calcarine fissure. Monakow claims that he has recent clinical and anatomical experimental proof to warrant him in saying that the visual area extends to the lateral surface of the occipital lobe.

The part which the angular gyrus (including the post-parietal) has in vision, is to subserve the purpose of secondary visual representation, or, to express it more colloquially, to serve as a storehouse of visual memories.

Lesion of the angular gyrus which does not involve or interrupt the continuity of the optic radiations, the subjacent optic fascicle, never causes hemianopsia, not even of a transitory character. A recent experience published by Déjerine fully corroborates this. The clinical manifestations were word blindness and agraphia. On autopsy the occipital convolutions, including the cuneus, were intact. The symptoms were produced by a circumscribed lesion in the parieto-occipital fissure, extending sufficiently deep to sever Gratiolet's visual radiation and the fibres which connect the visual perception centres with the centres for optical memories. Such a lesion does, however, cause word blindness. That is a condition in which words are seen, but their meaning, what they stand for, is not interpreted in the light of previous memories of such words.

In this connection it should be mentioned that a centre for elevation of the superior lid is placed in the angular gyrus by many investigators, especially by Grasset and Landouzy, and a number of corroboratory observations have been published; but there are so many cases in which ptosis has not been present and in which autopsies

showed extensive affection of the angular gyrus that the location of such a centre can only be admitted as decidedly problematical, if at all.

The existence of distinct centres for form, light, and color was claimed by Wilbrand on the ground that hemianopic color defects may exist together with normal fields for white, thus indicating the involvement of a color centre only; but Holden and others have claimed that corresponding light-sense disturbances also exist in every case, rendering this hypothesis untenable.

Auditory Localization.

The conclusions of physiologists and clinicians are not in entire accord as to the cortical localization of the auditory centre, yet it seems to be settled beyond question that the temporal lobe is the seat of the centre for audition. Of the physiologists, Luciani alone disagrees in part with this. He believes that simple auditory sensations are arrested in infracortical centres and interpreted in the intellectual sphere. Ferrier, Munk, and Forel concur in locating it in the temporal lobe, and their opinion is confirmed by the admirable work of Onuf (Onufrowicz). Schäfer and Brown concluded, from their extirpation experiments on monkeys, that the centre for audition is not in the temporal lobe; but Baginsky, from experiments on dogs, found that excitation of the lower part of the temporal lobe caused movements of the ear of the opposite side, which proves that the auditory area is not only a sensory, but a reflex motor centre for the ear. Ferrier, after a series of experiments instituted for this purpose alone, concludes that the views of Brown and Schäfer are untenable. As in the positive location of all the other centres, motor or sensory, the evidence based on the observations of physicians have been of greatest importance. Mills is of the opinion that the centre for the understanding of words is located in the posterior third of the first and second left temporal convolutions. Seppilli found in the brains of two deaf-mutes atrophy of the first temporal convolution. He concludes that audition is located particularly in the superior temporal convolution. It cannot be stated positively at the present writing whether or not the centre for audition is bilateral, or whether it is unilateral, like the motor speech centre. It is more than probable that in many individuals auditory perception is a bilateral one, and that one ear is served through the temporal lobe of the opposite side; but in order to have complete cortical deafness, it is necessary to have lesion on both sides, although interpretation of auditory impressions is unilateral and left-sided.

Destruction of the cortical auditory centre does not produce, apparently, crossed complete cortical deafness, but it does cause a marked diminution of hearing on the opposite side and a slight diminution on the same side. But even this, it seems, is not substantiated by some of the more recent clinical observations and experiments on animals.

We have learned from the brilliant series of studies on aphasia inaugurated by Wernicke and Kussmaul, in which word deafness, tone deafness, and other faults of sensory perception mediated through the ear, are discussed, that it is necessary to consider the auditory centre as made up of at least three parts: A centre for auditory perception, a centre for auditory memories, and a centre for the perception of words as words. Destruction of the centre for the last named gives rise to the condition known as word deafness. There is a wealth of clinical and pathological observation to show that this centre is unilateral, that it is situated on the left side for right-handed persons, and *vice versa*, and that its seat is the cortex of the posterior portion of the first temporal convolution. The seat of the acoustic perception of words, says Seppilli, is the left first temporal convolution; the other temporal convolutions of the same side are for the preservation of acoustic images indispensable to the expression of ideas. This hypothesis would seem to be borne out by a case of Cattani, in which there was verbal amnesia but no verbal deafness, and in which the lesion was found in the anterior portion of the left temporal lobe.

Brief reference must be made here to the speculations of Broadbent concerning the setting apart of a "naming" centre, because it has recently been made the subject of extensive consideration by Mills, in "Nervous Diseases by American Authors." Mills believes that an observation of a case, which he has made in connection with McConnell, justifies him in differentiating a naming centre in the temporal lobe. The patient had epileptic attacks, left lateral homonymous hemianopsia, word blindness, but not letter blindness, and could not name objects either from sight or from touch. On autopsy a tumor was found in the posterior and middle part of the third temporal convolution, the area which Mills believes should be referred to as the naming centre. It is to be noted that there was also a hemorrhage, reaching caudad as far as the white matter of the middle of the occipital lobe.

I am not inclined to concede the necessity of a separate naming centre, either from pure reasoning or from this observation. Moreover, it does not seem to me that the interpretation of this case is in keeping with our present knowledge of the genesis of speech.

We must probably assume here a destruction of the connections which subserve the associative complex which enters into word conception, and the complex of object conception. Destruction of such connections explains, for me, the inability to apply concrete terms to certain objects and things in this case.

Olfactory Localization.

The seat of the cortical centre for smell is not yet satisfactorily determined. The evidence at hand would seem to indicate that the uncinate gyrus is the centre of representation for this sense. It is necessary to state, however, that many contemporaneous writers subscribe to the views of P. Broca, to which he was led from work in comparative anatomy, and postulate the hippocampal convolution and the posterior part of the orbital lobule as the centre for smell. Clinical material illustrating disturbance of this sense caused by localized lesion is extremely rare. Experiments on animals to determine the location of this centre have not given uniform results, nor have they been conclusive. The location of the centre is probably such that it does not lend itself with facility to experimentation, and the pathological material heretofore has been slight and insignificant.

Experiments on animals, however, have shown that destruction of the centre, predicated as olfactory, causes disturbance of smell, predominantly on the same side as the lesion, and to a slight extent on the opposite side. From his original experiments Ferrier was led to believe that the cortical centres for olfaction were direct and uncrossed—that is, that the olfactory nerves went directly to the respective centre of the corresponding side. Most if not all later experimentation has gone to show that decussation of the intracentral project of the olfactory nerves does occur, but that the direct fibres are by far more numerous than the crossed.

It has been shown by Edinger, from experiments on osmatic mammalians, that the olfactory lobe and olfactory area are connected in various ways with the forebrain, the tweenbrain, and the midbrain. The olfactory bulb is connected with the cortex of the olfactory lobe, and with that of the cornu ammonis by means of the tangential fibre tracts, called the lateral olfactory radiations, and also by the deep medullary stratum which constitutes the mesal olfactory root and the olfactory bundle. The first passes in front of the olfactory bundle into the septum pellucidum; the olfactory bundle passes backwards after partial decussation into the fimbria of the cornu ammonis. Although the experiments on dogs and cats by Luciani and his pupils, particularly Fasola, led them to conclude that the cortical

centres of smell irradiate certainly in the parietal lobes, and that on the external surface of the hemispheres the contres are in the perisylvian convolutions, they recognize that destruction of the cornu ammonis causes distinct loss of smell, particularly on the same side as the ablation, and to a lesser extent on the other. One feature of Ferrier's findings concerning this centre, that offers excellent corroborative evidence, reasoning from analogy, is that electrical irritation of the uncinate gyrus causes muscular movements of the nostril, a form of movement analogous to that seen in the eyeball when the cuneus is stimulated, and in the ear when the first temporal is faradized, and to which the name reflex movement is given.

Their views are only partly in accord with those of Zuckerkandl, who believes that the entire limbic lobe is representative for this sense, for they teach that on the inner surface of this hemisphere the localization of the sense of smell is in the posterior part of the hippocampal convolution. In this latter contention they are more nearly in accord with clinico-pathological observations.

Griffith has recorded a case in which there was progressive anosmia in the right nostril, and after death there was found erosion of the right uncinate convolution, which was believed to have been the cause of the anosmia. The tip of the uncus was unaffected. Cases of epilepsy in which there is an olfactory aura preceding the attack are not extremely rare, although there are but few published records of autopsy of such cases. Hamilton has made a contribution of this kind, and so has Jackson. In the case of the first mentioned, softening of the temporal lobes was found; in the second, a tumor of the temporal lobe. Another interesting case has been recorded by Worcester, in which, aside from the epilepsy, the most important symptom was hallucination of smell. In this case autopsy showed a small spot of red softening in the left uncinate gyrus, in addition to a spot of softening in the temporal and in the frontal lobe. Frigerio has reported a case in which olfactory hallucination was thought to be due to atrophy of the cornu ammonis, which was found at autopsy. Mackey has recently published a case with cerebral tumor, in which loss of sense of taste may be referred probably to the softening found in the uncinate gyrus.

The relationship of the sense of smell to the sense of taste is a very intimate one, and in man the latter is complementary to the former. It is not a matter of surprise, therefore, that the frugal data at hand relative to the localization of the latter should indicate their close anatomical relationship—in fact, juxtaposition.

Gustatory Localization.

The alleged position of the centre for the sense of taste, at the base and on the internal surface of the hemisphere, is such that experimental evidence of its existence is difficult to obtain; while in man the sense is so closely associated with that of smell that it is difficult to obtain any pathological testimony of signal value. So far as evidence at hand goes, it indicates that cortical representations of the sense of taste are situated in the cortex of the inferior temporal convolution on its internal surface, just below the anterior termination of the gyrus hippocampus and its turned-up end, the gyrus uncinatus.

In this, as in the evolution of each of the other "centres," experimental physiology has given some ill-defined basis for clinical observation and post-mortem corroboration, which latter have always established or denied the right to what may be called autonomy. In almost every instance the centre suggested by experiment has been found too diffuse, and with very variable limitation. Bearing these facts in mind, it is possible that the experiments of Sentcherbach may be deserving of mention. Working on the hypothesis of Gad, that labial and buccal prehensile and masticatory movements, as well as those of swallowing, are "reflex," and that loss of voluntary power to perform these was always associated with ageusia, he determined the region of the brain in the rabbit destruction of which would cause these. He concludes that the gustatory centre is somewhat in front, and to a less distance behind where the cranial coronal fissure would impinge if extended into the substance of the brain.

Localization in the Centrum Semiovale.

Localization of lesions in the white substance of the brain cannot be made with anything approaching the accuracy of that of cortical localization. By way of introduction, it should be said that if the function of the centrum semiovale be kept in mind—that of serving the projection, commissural, and some of the association fibres with a passage way—it will be evident that lesion of this portion of the central nervous system will cause symptoms varying with the kind and number of fibres involved—that is, varying with the position and the extent of the lesion. The occurrence of lesion in this part of the brain is rare. This of itself would be sufficient reason to deter us from attempting to formulate any definite statements concerning localization of such lesion; but, in addition to this, there exists great

uncertainty concerning the relative course and position of many of the neural constituents.

To illustrate a form of symptom complex that may be produced by a lesion in the centrum semiovale, it is only necessary to refer to a case recently recorded by Wallenberg, in which there was complete left-sided paralysis of the facial nerve in all of its branches, paralysis of the left hypoglossal and left laryngeal nerves, and total paralysis of deglutition. Death occurred three weeks after onset of the symptoms, and was found to have been caused by a focus of softening in the right centrum semiovale, which, although comparatively small, had interrupted the following tracts of fibres: nearly all the projection fibres of the third frontal convolution, the ventral half of the second frontal gyrus, the annectant convolution of the third frontal, some of the constituents of the central convolutions, many of those from the lateral; and, in addition, the sagittal association tracts of the frontal lobe, and some fibres of the corpus callosum and of the external capsule. It had left untouched the caudate and lenticular nucleus and the internal capsule, all of which were ventral to the focus. The symptoms pointed so directly to involvement of the oblongata that during life the lesion was considered to be a bulbar thrombosis, limited to one side.

If a lesion of the centrum semiovale does not cause interruption of some tract of the projection fibres there may be no definite symptoms, as lesions of commissural and association fibres are not associated with a definite symptomatology. When disease of this portion of the cerebrum does occur, its location can only be suspected when lesions of other locations are excluded by the presence of some symptoms or complex of symptoms. The occurrence of psychical symptoms, such as alteration in the general mental state, is sometimes an evidence of lesion of this part of the brain, and is explained by the destruction of association fibres. In the same way are to be explained cases of psychical blindness which sometimes occur with such lesions. Motor symptoms are more apt to be present with pathological foci in this region of the brain than are sensory. In fact, monoplegias and aphemia are the commonest symptoms. When disturbance of sensibility does occur, the lesion is generally situated at a sufficient depth to involve the internal capsule in its posterior portion, and is apt to be associated with disturbance of vision and audition, because it involves in the *carrefour sensitif*, the projection fibres for sensory impulses and those for the higher senses.

It is self-evident that the nearer the internal capsule the lesion is situated the smaller it need be to cause pronounced symptoms, and the more liable it is to cause symptoms indicative of involvement of the capsule; while, on the other hand, the nearer it is to the cortex

the less liable is a small lesion to produce definite symptoms. Lesions of the centrum semiovale in its deeper parts are devoid of one symptom which is so suggestive of cortical implication, viz., epileptiform convulsions. These occur with lesion of the higher parts of the centrum semiovale only when the focus is immediately subcortical and impinges on the cortex.

Perhaps the most important diagnostic feature of lesions of the centrum semiovale are the variations in the clinical picture which they produce, from those caused by cortical foci on the one hand and capsular on the other.

Destruction of the corpus callosum is manifest by slowly progressing mental deviations, varying from mild delusions to attacks of mania; hemiparesis with contracture, and especially by paresis of one side, and slight paretic involvement of the other side; integrity of the cranial nerves and tendon jerks; and, if the destruction is due to a new growth, by the usual accompaniments of increased intracranial pressure in a moderate degree. Convulsions manifest on both sides of the body, but more severe on one side than the other, and not followed by paralysis, are also a frequent symptom.

Localization in the Corpora Quadrigemina.

Localization of lesion in the corpora quadrigemina presents no considerable difficulty, if there be no anomalous development of symptoms. The most common lesion of this part of the brain is tumor-growth. As Nothnagel pointed out a few years ago, the clinical picture develops with an unsteady gait, which is soon associated with ophthalmoplegia of both eyes, but not entirely symmetrical, and which does not involve all the muscles to the same degree, but manifests a predilection for the superior and inferior recti; all other symptoms are incidental or complications.

The disturbance of coördination is that commonly described as cerebellar ataxia, and is manifest both in station and in gait. In the upper extremities this ataxia sometimes resembles the athetoid movements and tremor of paralysis agitans (Ilberg). The ataxia is, however, increased on voluntary movement, intention tremor, and the movements are irregular, jerky, and choreic (Kolisch). The ataxia is in all probability not the result of destruction of a coördinating centre in the corpora quadrigemina—that is, it is not a local symptom due to destruction of the red nucleus or the decussation of the brachia conjunctiva, but is due to the interruption of coördination tracts which are directly and indirectly in connection with the quadrigeminal bodies.

The weight of opinion of recent writers on the subject of ataxia associated with disease of the corpora quadrigemina is that it is independent of and not associated with affection of the cerebellum, nor is it remotely operative through it. It must be stated, however, that it is entirely impossible to differentiate ataxia due to lesion of the corpora quadrigemina and that due to involvement of the cerebellum from consideration of it alone. With the cerebellar ataxia the condition of instability varies, but it is always present.

There are no pupillary symptoms of pathognomonic significance, but it is to be remarked that pupillary reaction is not disturbed in lesions of the posterior quadrigeminal bodies, and is lost with destruction of the anterior.

It is possible, also, that a consideration of the serial order of the development of symptoms may be of some service to distinguish lesion of the corpora quadrigemina from that of the cerebellum; ocular paralysis preceding the appearance of ataxia speaks in favor of lesion of the first named.

Attempts have been made to differentiate lesions of the anterior quadrigeminal bodies from those of the posterior, and it has been said that striking disturbance of vision is associated with lesion of the anterior quadrigeminal bodies; but there are no real grounds for such claims. In a recent communication, Weinland has called attention to the fact that disturbance of hearing is a frequent symptom with tumor of the region of the posterior quadrigeminal bodies. This is not at all surprising when we remember that the continuation of the striæ acousticæ, which forms the inferior fillet, passes close beneath the posterior quadrigeminal bodies. In one patient whose history is related there was very distinct diminution of hearing on the right side. This was found to be dependent upon tumor of the right and the posterior quadrigeminal region. In eighteen cases from the literature, disturbance of hearing was remarked in eight cases. In others auditory symptoms were not sought for. Involvement of the continuation of the striæ acousticæ in the fillet is probably responsible for the disturbance of hearing in some of these cases, and it is yet an open question whether lesion limited strictly to the posterior quadrigeminal bodies can cause auditory disturbance. Loss of pupillary light reflex and disturbance of vision, associated with the symptom complex already stated, point to location of the lesion in the anterior quadrigeminal bodies. Integrity of pupils, slight disturbance of vision, derangement of hearing, pronounced ataxia, in connection with an oculomotor symptom complex, point to lesion of the posterior quadrigeminal bodies.

It should be remembered that Bechterew cites experimental evi-

dence to prove that the posterior corpora bigemina are centres of hearing, of the voice, and of special forced movements and positions, *manège* movements. He does not admit that disturbance of phonation and other motor disturbance which follows ablation of these bodies is due to lesion of more deeply lying structures, such as the superior cerebellar peduncle, but he is convinced that the posterior bigemina are themselves centres for such movements, even despite the fact that superficial destruction of these masses is without effect.

Localization in the Peduncles.

The most important point to bear in mind in locating lesion in the peduncle is, that lesion of this structure produces motor paralysis of the facial and the hypoglossal nerves and of the extremities, which is manifest on the opposite side of the body. As in lesion of any other part of the nervous system, in which functionally different and important tracts are compressed into a relatively small area, the symptoms caused by lesion of the peduncle will vary with the size and extent of the lesion. Circumscribed lesion of the crusta alone may cause simple hemiplegia of the opposite side, without involvement of any of the cranial nerves or without other symptoms of any kind. In fact, cases have been recorded in which small lesions in the lateral part of the crusta, which left the middle third free, have produced no paralytic symptoms. This is explainable in accordance with the arrangement of the relative components of the pes pedunculi; the external fourth contains the fibres passing from the occipital and temporal lobes to the pons, while the next fourth is chiefly composed of the pyramidal tract; the next fourth is made up of fibres from the striate body, posterior part of the frontal convolutions, and fibres from the anterior central convolutions; and an internal fourth contains fibres from the insular region and the base of the lenticular nucleus, which end in the upper part of the pons. This last division is said by Flechsig to contain fibres from the frontal lobe, but this is denied by Zacher and other recent writers.

If the lesion is of sufficient size to encroach upon the sensory pathway of the peduncle, contralateral hemianæsthesia will be produced. More common than this, however, is crossed paralysis, partial or complete, of the eye muscles supplied by the third nerve, and hemiplegia—that is, paralysis of the oculomotor nerve on one side (the same side as the lesion)—and contralateral hemiplegia—a condition to which some recent French writers, in their zeal for eponymic nomenclature, have designated as the syndrome of Weber. When ophthalmoplegia results, it may be partial or it may be complete.

The former is more apt to occur from involvement of some of the nuclei of the oculomotor nerve, in the central gray matter of the aqueduct of Sylvius, as in interpeduncular lesion, although naturally such a lesion may produce ophthalmoplegia as well. The latter is often caused by peduncular and subpeduncular lesion, which involves the third nerve in the neighborhood of its emergence. Oftentimes when the ophthalmoplegia is incomplete, interpretation of the ocular symptoms is not easy. It will be facilitated, however, if it be remembered that the fibres of the third nerve, which supply the external muscles of the eye, take their origin from the distal end and middle third of the third nerve nucleus; while those for the internal ocular muscles, including accommodation and the pupils, come from the proximal division of the oculomotor nucleus.

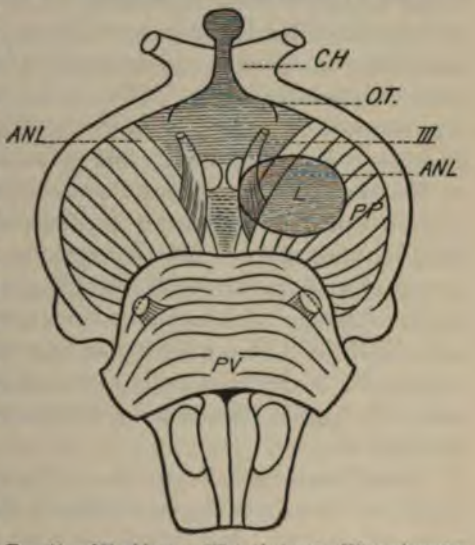

FIG. 11.—*CH*, chiasm; *OT*, optic tract; *III*, oculomotor nerve; *ANL*, ansa lenticularis; *PP*, pes pedunculi; *L*, lesion; *PV*, pons. (Brissaud.)

If ophthalmoplegia is associated with hemiplegia and hemianæsthesia, it may be stated positively that the lesion is within the peduncles. A subpeduncular lesion would rarely if ever be associated with hemianæsthesia. A subpeduncular lesion may be so extensive as to reach across the middle line in the interpeduncular space, and involve not only the third nerve of the same side just at its exit, but the nerve of the opposite side as well, and thus cause total or partial ophthalmoplegia of both sides, with hemiplegia of the side opposite to the involved peduncle. This is readily understood by reference to the adjoining figure.

In addition to this symptom complex, peduncular lesion is generally accompanied by vasomotor manifestations, sensations of heat and cold, paralytic perspiration, etc., on the side opposite to the affected peduncle. Whether ataxia and disturbance of the functions of the bladder and rectum, which are often accompanied by peduncular lesion, are essential, it is quite impossible to say. They are, at least, common accompaniments.

Localization in the Optic Thalamus.

Localization of lesion in the thalamus is very uncertain, as the functions and connections of this interbrain ganglion are not yet positively and accurately determined. It is more than probable that it contains a centre for mimic movements and secondary paths for laryngeal innervation (Eisenlohr). It has been thought that lesions of it are attended by subnormal temperature, but White from his experiments on rabbits concludes that lesions of the thalamus do not alter bodily temperature. Ott is led by experiments to believe that it is the seat of vasotonic centres. The fact that it is a regulator of motor acts has been completely established by v. Monakow.

Bechterew confirms Rethi's conclusions that the thalamic regions contain centres for chewing and swallowing and he thinks that they also contain centres for the various vegetative functions, such as movements of the stomach, œsophagus, and intestines, as well as for the involuntary movements participating in the expression of feeling and emotions. He believes also that in a certain way the thalami are centres for convulsive crying, and explains uncontrollable crying in cases of organic affection by interruption of cortical connections in the thalami.

According to Simbriger the optic thalamus occupies an intermediary and transitional place between the combination of sensory and motor functions, designated psychical, which is attributed to the cerebral hemispheres, with the preponderance of the one or the other, as the case may be, and the reflex function of all those parts of the central nervous system behind the optic thalamus, with the exception of the cerebellum. It possesses the ability to completely coördinate movements, to regulate all organic activities, particularly the nutrition balance and likewise those reflectory conditions which bespeak grades of consciousness and possession of will.

It confines itself to these functions in the lower animals devoid of cerebral hemispheres, such as the Amphioxus, to which it subserves psychical function, and it is the mediator between voluntary and reflex action. In all probability it has very much the same function in humans previous to birth, for in them the functions of the cerebral hemispheres develop very late. This is in keeping with the fact that in lower animals the optic lobes reach such a very great degree of development that they form the larger part of the brain.

Lesion of the thalamus is associated with contralateral hemiparesis, hemiataxia, loss of muscular strength, hyperæsthesia, amimia, involuntary crying, loss of coördination for the acts of eating, chew-

ing, and swallowing, and disturbance of vision in the shape of contralateral homonymous hemianopsia. It is very doubtful whether many of the symptoms, such as hemiparesis and hemianæsthesia, are due to lesion of the thalamus itself. In all probability they are the result of involvement of fibres of either the internal capsule or peduncle, although there is considerable evidence to show that contralateral anæsthesia may be due to thalamus lesion. One point to which attention should be directed is that lesions of the thalamus which have been reported to cause peculiar tremor, athetoid or hemichoreic movements, are not really the cause of such movements; such symptoms are attributable to lesion of the peduncle in the immediate vicinity.

Ecchymoses and even considerable hemorrhage of the gastrointestinal tract are not uncommon symptoms occurring in thalamus lesions, and have lately been noticed by Sinkler. Koenig was led to the conclusion from a study of cases of infantile cerebral palsy, in which paralysis of mimetic movements is common, that the connection between the thalamus and movements of emotional manifestation is not so intimate, or at least not so necessary, as is sometimes supposed. At least, it may be said that emotional control is not monopolized by the thalamus. He refers to a case in which there was paralysis of the mimetic face muscles, in which the thalami were found on post-mortem examination to be intact, while a circumscribed lesion of the motor area of the cortex was the only lesion to which the impairment of mimicry could be attributed.

Localization of Sensations Mediated through the Periphery of the Body, Particularly by the Skin, Mucous Membranes, and Muscles.

The difficulties surrounding a brief presentation of this subject are immense. In the first place a number of entirely different sensations are included under this heading, such as tactile sense, caloric sense, pain sense, pressure sense, position sense, motion sense or kinæsthetic sensation, not to speak of the so-called organic sensations. Most if not all of these are independent forms of sensation and not qualities one of another. Dermal sensibility is of paramount significance. It is the first sense that appears and it is of the greatest importance in contributing to the development of other forms of sensibility by processes of differentiation and selection. We shall confine our remarks to a consideration of the tactual sense and the muscular sense, as these two have been studied more carefully clinically and pathologically than any of the others, and as determination of their condition is without great difficulty by experimentation on animals. There

have been, and to a lesser extent there are to-day, two very diverging views concerning the location of tactual sense and muscle sense in the brain. The school of experimentists, on the one hand, headed by Ferrier, maintain that the gyrus fornicatus is the seat of their representation. Clinico-anatomists, on the other hand, with few exceptions deny the gyrus fornicatus such function and contend that the great preponderance of unassailable evidence points to the central convolutions, the cortical motor areas, as the principal area of representation for these sensations, and this is practically the claim of Munk and his school. The latter willingly admit that there is some evidence which cannot be entirely reconciled to their contention, but instead of allowing this to invalidate their conclusions, they seek some other explanation of this anomalous evidence.

Those who hold to the first view are not without the support of some clinicians. In this country Mills has unswervingly contended for the localization of what he calls general sensation, principally in the gyrus fornicatus and to a lesser extent in the hippocampal gyrus, the precuneus, and the postparietal convolutions, while in England cases have been reported which apparently indicate the doctrine of separate sensory localization. In the latter country the influence of Ferrier's personal teaching must always be reckoned with.

The experimental, anatomico-clinical, and purely pathological evidence pointing to the belief that the principal representation of tactual and muscular sense is in the motor areas of the brain becomes every year more conclusive, while the number of cases of injury to the cortex of the central convolutions without the occurrence of sensory disturbance gets less.

I do not wish to be understood as saying that the cortex of the motor regions and their immediate topographic surface vicinity is the sole seat of dermal and muscle sensory representation, nor that the confines of the area are as sharply delineated as those of the motor areas; but that it is the principal seat, and that the gyrus fornicatus, the gyrus hippocampus, and the postparietal convolutions have no more claim to be considered centres of sensory representation than any part of the brain that is the recipient of, or is responsive to, sensory impressions, seems to me the truth.

All of the most recent anatomical, clinical, and physiological evidence tends to show that central sensory localization, or the centre for dermal sensation, is in the motorial regions. There is much evidence to show that the centre for muscular feeling, *i.e.*, sense of position, sense of active and passive movements, is not only in the motor region, but also posterior to this region in the upper parietal lobules. This I believe to be the view supported by evidence which is con-

sidered scientific and which is reconcilable with the tenets of psychology.

It has before been mentioned that sensation mediated through the skin is the first to be developed and that the other functions of the brain are dependent upon its integrity for their development. The function of the brain that develops first after this form of sensibility is that of reflex and automatic action—which is dependent in the beginning, as it is at all time, on the former. Granting that these facts are so—and really they admit of no contradiction—it is hardly to be assumed that the principal central representation of this sensibility would be in a region remote from the cortical areas of motor representation, but rather in the closest anatomical association. It may be said that if an argument of this kind be used to substantiate the juxtaposition of the areas for motion and sensibility, one can as well say that the centres for peripheral sensibility are superimposed on those allotted to other senses. The supposition and conclusion would be alike erroneous, for other and higher forms of sensibility have developed from this simpler form by processes of differentiation and selection. It is believed, however, that the areas to which the more highly specialized senses are limited include a small representation of peripheral sensibility, and that movement in the peripheral organs through which these special senses are manifested, which experimenters get by irritating the respective areas with the faradic current, such as movement of the eyeballs by irritating the visual area, is to be explained largely, if not wholly, through this representation of peripheral sensibility. The most important experimental evidence tending to support the duality of function, motor and peripheral sensibility, of the Rolandic area of the cortex has been furnished by Mott, whose experiments have been corroborated to some extent by Schäfer and by Horsley. Mott found that in every instance excision of the motor area sufficient to cause monoplegia caused defective sensibility to all forms of stimulus. He states that in his opinion permanent defect of tactile sensibility results after large lesion of the motor cortex. Lisso states that cutaneous and muscular sensibility and motility are in the same region of the cortex. The first is situated superficially, the second deeper, and the last deeper still. He does not furnish any proof of his belief, nor does he state that this is practically the same claim that Nothnagel made on clinico-pathological, and Horsley on physiological grounds.

In his most recent contribution to the subject Dana has published four cases of his own and collected nineteen others from the literature, all of which go to show that lesion of the central convolutions causes defective peripheral sensibility. It would be unwise in a *résumé* of

the subject to cite all the individual cases that are on record in support of the contention that the motor region is the principal seat of peripheral sensory representation. Such cases have been recorded by Westphal, Seguin, Gray, Starr, Déjerine, and many others. The last-mentioned writer has recently maintained that motility, general sensibility, and muscular sense have one and the same cortical localization. He bases this opinion on the anatomical findings of a case in which cutaneous and muscular sensibility were impaired in proportion to motility. The fact that a small lesion of the motor cortex gives rise to no sensory defect and that complete destruction of one motor area does not produce absolute hemianæsthesia has been difficult to explain. We know that if the sensory tracts be severed in the posterior segment of the internal capsule complete hemianæsthesia will result, but that complete extirpation of one motor area produces only partial hemianæsthesia. Brissaud and Frankl-Hochwart believe that this can be explained by assuming that the sensory pathway goes largely to the opposite hemisphere, but partly through the corpus callosum to the other. It is difficult, however, to reconcile these suppositions with the apparently incontrovertible proof of its complete decussation furnished by Redlich.

Destruction of small areas of the motor region produces considerable motor paralysis and only slight sensory disturbance. The reason for this is readily apparent when we consider the origin of the motor tract from ganglion cells of the motor area. Each one of these is of individual importance and the beginning of an important neuraxon having a definite function to subserve, and destruction of it will cause loss of function in the structures supplied by the neuron. The termination of the sensory fibres, on the other hand, is by giving off collaterals and terminal arborization, and is not therefore confined to one small spot. A small lesion would scarcely include all the arborizations of a sensory neuron, therefore merely hypæsthesia follows limited or very circumscribed destruction of a cortical area.

Localization of Complex Mental Processes.

Function of the Frontal Lobes.

There are many facts, biological, physiological, anatomical, and pathological, that point to the frontal lobes as the seat of those neural elements whose functioning subserves the purposes of a physical basis for the higher or more complex processes of the mind. To the middle frontal and prefrontal regions have been allocated by different inves-

tigators centres of attention, of memory, of intelligence, of reasoning, of the higher psychical faculties generally, of apperception, and of character. There is some evidence of pathological origin supporting in a rough way these various localizations of special mental faculties, but again many facts contradict them, even so far as to indicate that the frontal lobes may be considered silent, latent, or functionless.

Direct stimulation of the cortex of this region is negative in result. Indeed Bianchi used the motor unresponsiveness of the frontal lobes to stimulation as a physiological means for delimiting their boundaries in the posterior direction. Ferrier reports movements of the trunk, head, and eyes, and Munk and Grosglik movements of the trunk and lower limbs from stimulation of this region. Fano believes that inhibition is the function of the frontal lobes. Luciani and Seppilli, Munk and Goltz are led to the belief from extirpation alone that the frontal lobes are a continuation of the motor zones. Ferrier, Mott, Schäfer, and others found by stimulation and extirpation experiments a centre for movements of the head at the root of the second frontal and for movement of the eyes in the posterior part of the same; while Bechterew and Mislawski, Lepine and Rochefontaine report that only increased secretion of saliva follows faradic irritation of these parts. Because of the association of eye and head movements with the bodily attitude of attention Ferrier considers the frontal lobes the centre of attention, and, assuming attention and inhibition to be identical, calls them the centres for direct inhibitory control over the lower motor centres. Partial or complete ablation of the frontal lobes has been reported to have diverse consequences. Munk, Horsley, Schäfer, Grosglik believe there is here a centre for movement of trunk or a "*Fühlsphäre*" and as such a continuation of the parietal lobes. It is certain that removal of these areas produces no sensory disturbances or at least only slight, and for the most part temporary ones; that movement is slightly impaired manifesting itself chiefly as difficulty in coördination for complex movements (climbing, picking objects from floor—Bianchi), or in head and eyes (Ferrier), or in trunk (Grosglik, Munk). The very careful experiments of Bianchi show in addition to these minor symptoms a considerable deterioration in mental and emotional manifestations of a complex sort. A monkey which is affectionate, concupiscent, inquisitive, active, attentive, distinguishing between a threat with a stick which is only a feint and one that is real, after removal of the frontal lobes becomes inert, shows some forced movements, exhibits weakness of attention, is moved to excessive reaction of fear by a threatening gesture or by a noise of no great intensity or by the presence of its keeper, loses its inquisitiveness and affection for its keeper, is unable to distin-

guish clearly between a bit of sugar and a piece of plaster, is unresponsive to sexual stimulation, or exhibits an unwonted cruelty towards other animals of its kind.

Pathological evidence corroborates in part these experimental results of Bianchi. The American crowbar case is a famous instance. The patient lived thirteen years after having had a crowbar driven through the left frontal lobe, but his mental condition was one of childishness, his moral tone was diminished, and he was subject to epileptic fits. Lepine mentions intellectual dulness along with other symptoms, in one case with disease of the frontal lobes; in another, cerebral excitation, ambitious ideas, enfeebled memory, imperfect speech, bizarre character. Welt speaks of changes in character and disposition. The patient became malicious, slanderous, violent, quarrelsome, and dirty after traumatic injury to the frontal lobes. Abundo mentions lessening of perception, memory, and intelligence, and impairment of intellectual functions, occurring after injury to the frontal lobes.

Mills says that disease of the frontal lobes causes psychical disturbances which the physician is frequently not well enough trained to study—mental slowness and uncertainty, want of attention and control, impairment of judgment and reason. Inhibitory influence, both psychical and physical, is diminished. Memory is not seriously impaired, although a continuous train of thought cannot well be followed and complex intellectual processes cannot be thoroughly performed.

Gowers writes that in some cases considerable mental change has been observed, various changes in character, but sufficiently frequent and considerable to be of significance. A large number of cases, in which psychical disturbance was the only symptom, and greatest when both frontal lobes were injured, is found in neurological literature.

The frontal lobes have remained so long a *terra incognita* to the localist largely because of psychological misconceptions of the condition and analysis of mental processes. Changes in character and mental ability, which may amount to considerable in the life conduct of an individual, are difficult to ascertain in the course of a few observations and next to impossible to determine in the lower animals. The common understanding still looks upon the human mind as divisible into compartments—the so-called mental faculties—and seeks for certain specialized centres of attention, of memory, of association, of imagination, of æsthetic sense, of moral judgment, of will, of character, or even of the special talents of the painter, musician, mathematician, or logician.

When Wundt has been unable to express himself with sufficient clearness to avoid such diverse interpretations as have been given to his exposition by psychologists as prominent as Ward, James, and Münsterberg, it is hardly possible for me to put in small compass the psychological attitude that should guide the cerebral localist in positing to parts of the nervous system functions that are the physical basis of complex mental processes. It should be borne in mind, however, that modern psychology views all mental faculties as arising from a complicated coördination or adjustment of movement to the environment. Present and past experience act together upon the human organism to bring out motorial response in accordance with the life habits or impulses or instincts of the organism. Memory is fundamentally based upon the possibility of modification of cell structure or function through external physical or internal physiological stimulation.

Memories of the past environments presented originally through the sense organs as perceptions are evidences of the revival through central stimulation of an activity of the cells, whose function was initially excited by the various sensory impulses. Succession of such activities of revival is the physical basis of a train of association or thought. Variations in the succession and complexity of such associations give rise to organized memory, to imagination, either reproductive or productive, and to the specialized thought habits or talents of an artist, musician, or scientist.

The study of the development of the moral nature of an individual presents us with a picture of old habits of reaction broken up and modified and with the formation of new habits of action and thought. We do not therefore look for a specified area for the moral sense, for imagination, or for memory. The same cells that gave an immoral act may give a moral one. But we do look for regions that may subserve the purpose of a more complex coördination or adjustment than is found in the centres of the cord, or basal ganglia, or even in sensorimotor centres of speech.

The emotions are looked upon as conscious states conditioned by bodily reactions widely distributed throughout the organism, but all adding a sensory contribution to the activity of the cells of the cortex. Thus an emotion has no locality, but is the result of the sensory coördination of a number of nerve elements in the cortex. The feeling of agreeableness and disagreeableness or even of pleasure and pain has been analogously analyzed by Münsterberg: the one as due to the cerebral effect produced by a reflexly excited movement of general extension, the other by a similarly excited movement of flexion (*cf.* article on Pain in a later volume of this series). Attention, which

Ward and Ferrier regard as the fundamental element or condition of all mentality, is an adjustment of the organism (psychophysical) to a particular stimulus, object, or idea. It is a complex adjustment involving more of the organism, but nevertheless is typified by the reflex adjustment of lens, iris, and ocular muscles to a visual object exciting originally only the retina or a part of the retina, but which tends to call out such motor adjustments that the original exciting stimulus gains the position of visual advantage in the centre of the conscious field and thus is attended to.

The mental constitution of a personality, or of the character of an individual, or of apperception may be similarly conceived as the resultant of the adjustment of the whole conscious organism to an object of the environment or of thought.

In the development of these complex habits of movement, of feeling, and of thought, and for their continuous persistence, some portions of the central nervous system may be more essential than others. The frontal lobes may furnish an important cell basis for these coordinations. And yet other parts may be of importance too, and lesions elsewhere may cause a serious mental disturbance in coordination as do lesions of the frontal lobes. The left brain contains the centres of speech and writing because the organism is right-handed; but these centres may develop on the right side and under favorable external conditions they may separate, one developing on the right side and the other on the left. That injuries to the frontal lobes in some cases produce no apparent diminution in mental ability may be due to the fact that lower centres have acquired the automatic capability of maintaining these acquired coördinations. There is evidence that reflexes or automatic action may be continued without the higher centres that were essential for their origination. Normal use is necessary for the development of cerebral function. Congenital blindness is accompanied by absence of visual ideas of imagination, of color, and of visual dreams. On the other hand, many cases of blindness acquired after five years of age present us with powers of color imagination and conception and with vivid visual dreams. These phenomena frequently diminish with the continued blindness of the patient.

Thus, although there is no centre of attention or thought or apperception or morality in the frontal lobes, nor elsewhere in the brain, yet the frontal lobes have been shown to contain neural elements whose integrity must be maintained if the individual is to develop and persist in habits of attention, concentration of thought, balance of feeling, sound judgment, and moral conduct.

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ENCEPHALITIS.

Introduction.

Progress in the understanding of nervous diseases is nowhere more strikingly manifest than in the comprehension of the disease or diseases included under the term encephalitis. I do not mean to infer that much confusion does not still exist regarding the occurrence; the classification, the course, and the phenomena of these diseases, but, compared even with two decades ago, when Huguenin wrote his famous article on this subject in Ziemssen's Cyclopædia, our understanding of the matter is immeasurably clearer. By the term encephalitis inflammation of the brain substance is meant. It is also sometimes spoken of as cerebritis. The former term is to be preferred, as the inflammatory process need not by any means be limited to the cerebrum. Much confusion has resulted from failure on the part of clinicians and pathologists to concede that the same patholog-

ical processes may go on in the brain tissue as in any other tissue or organ of the body. It is to this that we must attribute so much of the obscurity that still clings to the various forms of softening—red, yellow, white—which are said to occur in the brain. As a matter of fact these conditions do occur, that is, they are found after death, but they are associated with a great variety of pathological processes and do not by any means follow *seriatim* as sequences of inflammation.

The forms of softening which occur in the brain are as various as are the forms of destruction of tissue that occur in the liver in the various diseases to which it is subject. To say that they are all inflammatory is absurd; and yet it was to the persistent advocacy of this view, particularly by Durand-Fardel and his followers, that so much confusion in the conception of these conditions of the brain was due. Even after learned pathologists such as Virchow and Hasse had shown unequivocally the untenability of this view, it still found adherents.

It is believed that our knowledge of pathology to-day justifies us in adopting the following classification or subdivision of encephalitis: 1. Acute, non-purulent encephalitis. 2. Purulent encephalitis. 3. Interstitial or chronic encephalitis.

Acute non-purulent encephalitis in the majority of cases is undoubtedly hemorrhagic, and it therefore merits this additional descriptive term. It is commonly described as acute hemorrhagic encephalitis or acute primary hemorrhagic encephalitis in order to emphasize the fact that previous change has not existed in the blood-vessel. At least, acute encephalitis must include a form in which the hemorrhagic element is the preponderant one, pathologically speaking. This, of course, is a difficult statement to substantiate. First of all because these cases, as has been shown by Oppenheim, ordinarily terminate in recovery without consecutive manifestations or sequelæ, and secondly because the structure of the blood-vessels of the brain is such that no considerable amount of inflammatory process can take place within the walls of the finer and more delicate ones without leading to rupture. Under the second division are classified those forms of inflammation of the brain substance which terminate in the formation of pus. As in all other tissues these processes are circumscribed or diffuse; if the former it is known as abscess, if the latter as purulent infiltration. Under the third division of interstitial encephalitis are included a part at least, and we believe a considerable part, of the cases heretofore described as cases of general and diffuse sclerosis. It will be seen in the description of that disease that Uthoff has placed this contention beyond cavil. Chronic encephalitis also includes some cases of atrophy of the brain,

a portion of the spastic diplegias, and other forms of cerebral infantile paralysis.

In endeavoring to bring order out of the chaos into which the subject of inflammation of the brain has fallen, many authors, in contributing to the subject, have given different names to the disease. These we believe can be reconciled by this classification. Poliencephalitis is the name given by Strümpell to inflammation of the gray matter of the cerebral cortex. He limits the application of the term to the pathological condition which forms the basis of a form of inflammatory infantile cerebral paralysis of non-traumatic and hemorrhagic origin, which he considers to be analogous to poliomyelitis. The name of Wernicke has been associated eponymically with a symptom complex known as polioencephalitis hæmorrhagica superior—a disease in which the lesion is concentrated in the central gray matter of the third ventricle, occasionally extending through the gray matter around the fourth ventricle even to its lower end and into the spinal cord, the collections of ganglionic cells in these regions being principally if not primarily involved. Poliomyelencephalitis, inflammation of the gray matter of the brain and spinal cord, is a term that has been much abused. It has been applied to conditions in which the lesion has been where the name indicates, that is, to a further extension of the lesion on which Wernicke's syndrome is dependent, and, on the other hand, it has been applied to cases in which the symptom complex has pointed the lesion to be in that locality but in which after death no lesion has been found. The latter use has been made particularly by Dreschfeld. It remains to be said that inflammation of the brain substance may limit itself to a particular portion of the brain; that it may be diffuse and widespread; that it may seemingly involve structures which are anatomically associated or the functions of which may hang one upon the other; and lastly, that symptoms pointing to more or less diffuse inflammation of the brain, the cortex above and the central gray matter lower down, may occur without the presence of detectable post-mortem conditions.

Softening of the brain, as has been said, is a part of all of these inflammatory processes; it is not, however, an essential part. In cases of encephalitis which recover there may be softening. In all the other forms softening is certainly never lacking. The so-called red softening occurs when the substance of the brain is the seat of profound inflammation. Yellow softening occurs in purulent encephalitis. White softening is nothing more or less than gradual slowly developing encephalomalacia, a condition which, as will be seen later, is associated with a variety of causes. It is not consistent with the present teachings of the pathology of inflammation to say

that true encephalitic processes can occur in the vicinity of neoplasms and of other collections in the brain which excite pressure, unless such collections be of microbic origin. When they are otherwise, the process that goes on about such a new formation is a truly degenerative one, the gross result of which may be white softening. Likewise the changes that go on around infarcts, unless the latter be of pyogenous origin, are degenerative and not truly inflammatory, although the process may be an acute one, just as the tissues of the brain which are the seat of true encephalitic processes may be surrounded by areas of degeneration.

It is undoubtedly a fact that many cases which were described by the older records as cases of meningitis, brain fever, and perhaps some of those described as typhus were true cases of encephalitis. Such cases are recorded in the writings of Wilks and Moxon and in that great storehouse of information, the works of Abercrombie. And it is no less a fact that many of the cases described by writers of to-day under the name of meningoencephalitis are cases of the latter disease, and especially of the hemorrhagic form. It is a fallacy, difficult to overthrow, that cases of inflammation of the brain substance, even of the cortical substance, do not exist without coincident inflammation of the meninges. Such a position cannot be held to-day in the face of the many authentic observations in which inflammation of the cortex has occurred without such concurrence. That we may not be misunderstood, however, it is necessary to say with emphasis that encephalitis may, and does often occur secondary to the various forms of meningitis, be they traumatic, septic, tuberculous, or post-infectious. It is manifestly impossible to give a description of an acute encephalitis whose pathological basis is neither hemorrhagic nor purulent, although theoretically such a condition may exist and cases which eventuate in recovery postulate its existence, because we have no anatomical findings to corroborate its occurrence.

Acute Hemorrhagic Encephalitis.

Acute hemorrhagic encephalitis is a condition whose anatomical basis has latterly been very carefully studied and substantiated. It is particularly to Strümpell, Fürbringer, Leichtenstern, Oppenheim, and to their pupils that we owe the elevation of this disease to a separate, well-defined entity. It is due to the fact, as will be shown under prognosis, that so many of these cases terminate in recovery, that we have not been in possession of a fuller knowledge of their pathology and morbid anatomy.

Etiology.

The preponderating consensus of opinion regarding the causation of this disease is that it is dependent upon some infectious process, and that the most important element in this infection is either the bacillus of influenza, whether that be the organism described by Pfeiffer, by Cannon, or one which yet remains to be discovered, or the pneumococcus. This is in keeping with the fact that the most valuable additions to our knowledge of this disease have been made since the influenza prevailed epidemically at the beginning of the present decade. Although the organisms are not always found after death, there are not lacking cases in which they have been found (Pfuhl, Nauwerck). Another and an important corroborative evidence is the fact that many cases of hemorrhagic encephalitis occur during an epidemic of influenza in patients who have none of the ordinary accompaniments of the latter disease. The same may be said of pneumonia, for it is with cases of influenzal pneumonia that hemorrhagic encephalitis has most frequently occurred (Baginsky). Whether such influenzal encephalitis results from the absorption of toxins from the pulmonary lesions, from infection, or directly from the local action of the bacillus cannot be said. It occurs also in connection with other infectious diseases, particularly epidemic cerebrospinal meningitis, and the infectious fevers, such as typhoid and typhus. In a number of cases it has been seen sequentially to malignant or ulcerative endocarditis. It may occur as a primary affection, as in the cases reported by Strümpell, Bucklers, Koenigsdorf, Schmidt, and others. In these cases the writers would have us believe, and I hold their contention not an improbable one, that the disease is an infectious one, pure and simple, and that the striking manifestations of the infection are the changes in the cortex. It occurs also in conjunction with the puerperal state (Patru), although it is a serious error to describe areas of hemorrhagic softening which occur after sinus and venous thrombosis as multiple hemorrhagic encephalitis, as has been done by Patru. It has been seen complicating epidemic cerebrospinal meningitis, acute superior polioencephalitis, or, we might better say, preceding it. It occurs most frequently in the young, that is, under twenty, and more often in females than in males. Putnam has recorded its occurrence in a patient seventy-one years old. It is not improbable that many of the cases of post-infectious "brain fever" occurring after measles, scarlatina, small-pox, etc., are cases of encephalitis. Oppenheim has noted its occurrence in a patient suffering from anæmia. It is probable that trauma has no etiological

significance, and to describe concussion of the brain and meningeal hemorrhage as a case of traumatic encephalitis, as Rendu has recently done, is absurd. I have seen instances of this disease, or at least of conditions with a similar complex of symptoms occur after prostration from heat. It may occur coexistingly with inflammation of the brain coverings, and thrombus of the sinuses and abscess of the brain (Virchow, Senator). Köster is of the opinion that he has seen cases which were due to erythema nodosum.

Symptomatology.

The disease develops as a rule without premonitory symptoms, or if such premonitory symptoms occur, they are frequently overlooked. This may be due in part to its occurrence in children. Sometimes the advent of symptoms is so abrupt and striking that the patient lapses at once into a stuporous condition, which quickly deepens into coma. Ordinarily headache of considerable violence, fever, reaching to 105° F., which may or may not have been preceded by chills, vertigo, vomiting, irritability, photophobia, and delirium, are the initial symptoms. In a case reported by Bucklers the disease ran its course without fever. Fränkel has also reported cases which ran an afebrile course. The advent of these is quickly followed by a state of mental hebetude, vacuity, stupor, and by manifestations of impairment and abolition of function of the part or parts of the brain involved. These symptoms coincide with the stage of exudation and pressure in the brain, so, naturally, the phenomena are paralytic. Symptoms of meningeal irritation such as stiffness of the neck, pin-point pupils, and hyperæsthesia are not present. The condition of mental hebetude is not so deep that the patient cannot be aroused to some slight degree of mental activity, but if partially aroused, he quickly relapses. The respirations are shallow and frequent, and later in the disease they assume the Cheyne-Stokes type. The pulse is rapid and becomes progressively feeble, it lacks the tense, resistant character of meningitis. The pupils are generally equal and respond to light. The deep reflexes are diminished though frequently superficial reflexes are present. In the beginning the sphincters preserve their function, later in the disease they may be inhibited. Other concomitants of acute infectious disease, such as enlargement of the spleen, have been noted in a few cases. In rare instances the onset of the disease is so very abrupt that when the patient is first seen by the physician he is already in a state of profound unconsciousness. Such a case has been reported by Bucklers. In others the course of the disease is a slow one and is character-

ized by severe headache, loss of appetite, severe thirst, and feelings of heat in the head. Very frequently some form of aphasia is an early symptom in these cases, even before the more severe cerebral symptoms develop. In many cases this aphasia is one of the most prominent symptoms. In three cases reported by Oppenheim it developed about ten days after the beginning of the disease. It is of the character of motor aphasia and paraphasia. In some instances optic neuritis develops first in one eye and then in the other, more rarely in both simultaneously. Hemianopsia was made out in one of Fürbringer's cases. Although the disease may run its course without the development of motor or paralytic symptoms, their occurrence is the rule. They may consist of monoplegias or hemiplegias, partial or complete, and in some instances of even more extensive paralysis, even to complete paralysis of all four extremities (Fürbringer). These motor paralyses do not come on abruptly as after cerebral hemorrhage or encephalomalacia, but are of gradual development. The motor impairment may be of no greater intensity than to cause paresis. Generally the patient makes no complaint, but does not like to be disturbed. Percussion of the head is resented. In those cases in which there is no loss of consciousness and tests for sensibility can be made, this part of the nervous system is found intact. Evidences of motorial irritation, such as spasms or convulsions, are uncommon phenomena of the disease, although an epileptoid paroxysm may usher in the symptoms of the affection. The course of the disease may be characterized by a progressive increase in the severity of the symptoms and finally the disease eventuates in death; nevertheless remission not infrequently takes place, that is, of course, remissions in the severity of the symptoms, more of the general than of the focal symptoms. In this way, after the patient has been in a stuporous, almost unconscious asthenic condition, he may pass for some time into a wakeful, restless, erethistic state.

If the seat of the hemorrhagic, inflammatory condition be in the pons, the oblongata, or the cerebellum, focal symptoms more or less characteristic of diffuse lesion of these parts, in addition to those already described, or apart from them, will be the result. Several cases of acute inflammation of the regions have been reported as developing after influenza. If in addition to the symptoms which have already been described as occurring in acute encephalitis there be optic neuritis of early occurrence, hemiataxia, marked disturbance of orientation, nystagmus, etc., involvement of the cerebellum is strongly suggested. If paralysis of the ocular and other cranial nerves is an early concomitant of the disease, and particularly if it occurs abruptly, it is probable that the brain substance at the

base is the seat of the encephalitic lesion. The symptoms characteristic of or occurring with poliomyelitis bulbi are enumerated in the chapter on diseases of the oblongata.

Course and Prognosis.

The course of the disease in the majority of cases is not uniformly progressive. It is characterized by periods of exacerbation and remission. In this way the duration of the disease may extend over a period of from many weeks to many months; that is, it may develop into a form of disease to which the name chronic is truly admissible. To Oppenheim more than to any one else are we indebted for pointing out that acute hemorrhagic encephalitis is not a disease so inimical to life and health as was formerly considered. He has reported a number of cases, as have others before and after him, in which complete recovery has resulted. Nevertheless the prognosis is always grave and depends largely upon the mode of onset, the severity of the symptoms, and the continuance of the latter without remissions, but more on the cause of the disease than everything else. Very abrupt onset, high temperature, and intense focal symptoms are signs of danger, while a slow onset, low temperature, protracted course, and absence of severe focal symptoms are of good omen.

The disease is to be differentiated from acute cerebral and cerebrospinal meningitis, from cerebral hemorrhage and from disseminated sclerosis with acute symptoms, and sometimes from brain tumor. The absence of motorial irritation, the early appearance of focal symptoms, such as paralysis and aphasia, the early occurrence of hebetude and loss of consciousness, the absence of projectile vomiting and hard, tense, wiry pulse speak against a diagnosis of the former; yet it should be remembered that in many instances the diagnosis cannot be made (v. Jaksch). According to Fürbringer a differential diagnosis between acute non-purulent encephalitis and certain cases of cerebrospinal meningitis is impossible. The period of life at which it most often occurs, the absence of any premonitory symptoms indicating disease of the vascular apparatus, the onset of symptoms after some acute infectious disease, such as influenza and the more slowly developing focal symptoms, combined with a less profound alteration of consciousness will differentiate it from the symptom complex of the cerebral apoplexies. It is not with the more acute form of encephalitis that disseminated sclerosis is apt to be confounded, but with those cases that pass into a chronic condition. The points in common of these two diseases will be discussed under the latter.

Pathology and Morbid Anatomy.

The essential pathological condition is an acute inflammatory process in the blood-vessels of the brain; the predominant features being small hemorrhages and capillary emboli, varying in size from a point so small as to be detected only by the microscope up to punctate extravasations of considerable size. These may occur in different parts of the brain. In some cases, such as those described by Strümpell and Leichtenstern, the motor area of the cortex seemed to be the favorite seat of the lesion. Not infrequently the areas of inflammation are developed in symmetrical parts of the brain. The centrum semiovale, the temporal lobes, the base of the brain, and the corpus striatum may be the localities of most profound involvement.

The common pathological changes are, in brief, vascular hyperæmia, hemorrhagic exudation, leucocytal infiltration, and retrogressive changes which lead up to softening or a cessation of these processes and a gradual return to the normal state.

To the naked eye the inflamed area is red and swollen, the normal differentiation between the gross constituents of the brain matter is obscured, and to the touch the tissue is less resistant and in some cases, depending upon the intensity of the inflammatory process, is almost pultaceous. Under the microscope the blood-vessels are seen to be distended and overladen, particularly the smaller ones, many of which are ruptured, the contents of the vessel—the red and white corpuscles—being found in the surrounding tissue. The lymph spaces are distended, and if the disease has been of long standing there will be seen an increase of granule cells and a more or less intense proliferation of the glia substance. Goodall has shown that the effect of experimentally produced encephalitis was a marked change in the spider or scavenger cells, the so-called cells of Deiter. These cells undergo not alone increase in size, but their nuclei and bodies take up very much deeper staining, and their processes, including the vascular, became very prominent. These changes occur earliest and most intensely in those regions in which the cells are most conspicuous in health, in the subcortical region. Whether or not these findings will be corroborated by their occurrence in pathological cases, cannot be said. If the inflammatory process has been a severe one and particularly if it be of long duration, the nervous elements must suffer up to the point of destruction. But, as in many cases complete recovery results, it is not probable that the latter is often the case.

If the disease occurs concomitantly or associated with other infec-

tious processes such as meningitis, thrombosis of the lateral sinus, etc., as it sometimes does, the morbid conditions natural to these diseases will be present.

A most interesting assertion has been made by Hasse, to the effect that the cicatricial foci remaining after encephalitis may be the starting-point of a new encephalitic process, and that tumors may take their origin from the seat of, and perhaps be in some way influenced in their development by, these former inflammatory areas.

A rich field for observation is open in the cases of this disease that terminate fatally, for the application of the Nissl stain and the Marchi-Algeri method of staining. It is very probable that the former will reveal changes in the dendrites and protoplasmic prolongations of most of the ganglionic cells which are surrounded by, or in the immediate vicinity of the hemorrhagic, inflammatory processes. It may also reveal the amount of injury which such cells will stand and still go on to recuperation. Theoretically, the latter method should reveal, by deposition of granules in the neuron pathways caudad to the seat of the lesion, destruction just in proportion to the intensity, severity, and location of the lesion.

Treatment.

The treatment does not differ essentially from that applicable to meningitis. After securing absolute rest and freedom from all disturbing factors, the comfort of the patient will best be contributed to by the administration of a dose of calomel, followed by a promptly and briskly acting saline, while the head is enveloped by ice bags. The diet should be of the blandest, most assimilable kind. If the patient be vigorous and robust, and particularly if the symptoms be of asthenic variety, the calomel may be preceded by local bleedings, by wet-cups, or by the application of leeches. Compression symptoms may be relieved by lumbar puncture (see article on Meningitis). If the patient's physical condition has been weakened by a previous acute disease such as influenza, these severer measures are not justifiable. Morphine should never be given. The intense headache and the elevation of temperature are best combated by the administration of small doses of bromide, by antipyrin, the salts of salicylic acid, and possibly by the administration of quinine. As the latter physiologically causes hyperæmia of the brain, vertigo, etc., it should not be given in very large doses and but tentatively even in small doses. The internal administration of mercury and of iodide of potassium, on the antiphlogistic theory, can be of only slight use. If the disease passes into a subacute or more or less chronic condition, counter-irri-

tation of the skull over such parts of the brain as the symptoms indicate to be the seat of the disease, and the internal administration of iodide of potassium are recommended. Accompanying or secondary diseases such as thrombosis of the lateral sinus and abscess, which sometimes develop, are to be treated in the usual way, surgically.

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Brain Abscess—Purulent Encephalitis.

When inflammation of the brain substance is caused by pyogenic organisms, the result is a purulent encephalitis. If the inflammatory process is circumscribed and the collection of pus limited, it is called brain abscess. If it is not limited it should be called diffuse

purulent infiltration. Clinically these two conditions are not separable. Formerly the pathological and anatomical conditions now described under brain abscess were referred to when seen at the autopsy table as red softening. The name red softening now, however, is rarely used, and when used has scarcely any pathological significance, as it represents but a stage in a number of conditions arising from as many different causes.

Of all the diseases to which the brain is liable, abscess of the brain is one of the most, if not the most, important. Its causation is reasonably well known; its pathology is clearly understood; and its treatment calls for but one measure. This knowledge calls for the expenditure of time and great care to insure the diagnosis in order that early measures may be adopted both for the prevention and cure of the disease. Until a few years ago abscess of the brain was considered one of the rarities of an individual experience. The decade now passed has shown that abscess of the brain is, next to meningitis, one of the commonest intracranial diseases. Hand-in-hand with the wider recognition of the disease has developed the power to cope with it. Ten years ago the hopelessness of cases of brain abscess was appalling. To-day, if skilfully handled, surgical treatment gives far greater prospect of relief than in any other intracranial lesion.

Etiology.

The causes of abscess of the brain are direct and indirect. The direct causes are the pyogenic organisms. Under these may be included the staphylococcus pyogenes aureus, the streptococcus pyogenes, the gonococcus, the diplococcus pneumoniæ, the bacterium coli commune, the tubercle bacillus, actinomyces, and very rarely the bacillus of typhoid fever. In two cases the parasite of oidium albicans was the direct attributable cause. Of these the two first mentioned are by far the most important. When they are present in a certain degree of intensity, particularly if the nutrition of the brain be lowered for any reason, and the resisting force of the tissues be weakened, they almost invariably produce brain abscess.

The indirect causes of brain abscess, although they would be inoperative if the direct causes were not present, are of great importance. First because they are the elements from which the pyogenic organisms set out for the brain, or they are the factors contributory to the entrance of the pyogenic organisms into the brain. These indirect causes are trauma; suppurative disease of the middle ear, or suppurative process in any of the cavities or sinuses of the skull, such as the naso-pharynx, which are in close communication,

directly or indirectly, with the contents of the skull; purulent disease of the frontal and ethmoidal sinuses, the orbital cavities, or others of the brain sinuses. In a considerable proportion of cases the pyogenic organisms come from a distance and have their beginning in bronchial dilatation, chronic pneumonia, pulmonary phthisis, empyema, gangrene of the lungs, septic endocarditis, etc., which therefore become indirect causes. Brain abscess is one of the rarest complications of the general septic state, under which is included pyæmia and septicæmia. It has not been seen so commonly as a sequel of influenza as has the previous form of encephalitis. The frequency with which brain abscess follows chronic suppurative disease of the middle ear has been variously estimated. The consensus of opinion, however, of neurologists and otologists is that about thirty-five per cent. of all cases are traceable to this affection. Considering the curability of chronic otitis media this is either a reflection on the skill of the otologist or an indication of failure on the part of the general practitioner to point out the danger of such aural conditions to parents. Acute purulent diseases of the middle ear do not seem to hold a very important causative relationship to brain abscesses, the chronic form causing brain disease about six times more frequently. Usually the aural condition is one of several years' standing and of but little trouble to the patient. In fact, in some cases in which brain abscess has been the result of this disease, the sufferer has completely forgotten the existence of previous or present ear disease. The relative proportion of ear diseases to brain abscess has been stated; it does not come within our province to estimate the ratio existing between brain abscess and diseases of the ear except to say that in our opinion the statistics given by Jansen cannot be taken as a standard without further corroboration. He studied 13,000 patients in the aural clinic at Berlin and found 6 cases of brain abscess. Of these 13,000 cases over 5,000 were cases of suppuration in the middle ear. According to his statistics, the ratio between brain abscess and suppuration in the middle ear is as 1 to 500 in chronic otitis, and as 1 to 2,650 in the acute form. Brain abscess dependent upon ear disease occurs almost always in infancy and in early adult life, and the most common causes of the suppurative ear trouble are the acute infectious diseases. It may or may not be associated with caries of the bony structure, in which the middle ear is situated; or it may in some cases have given rise first to septic inflammation in the lateral sinus, the brain abscess being then secondary to that.

Trauma, the next most important indirect cause of brain abscess, is responsible for a large number of cases. It would seem from a careful study of the reported cases of brain abscess that trauma may

act in more ways than one to cause brain abscess. For instance, direct trauma to the cephalic extremity may produce an open wound which exposes the outer table of the skull and this may be the direct pathway of the pyogenic organisms coming from without to the brain. These are the cases of brain abscess that develop within a comparatively short time after injury, and are mediated by extradural inflammation, possibly purulent in character; or the trauma may set up at the seat of injury changes in the tissues, which facilitate purulent formation, and those remaining may be the *loci* from which the septic organisms pass to the brain through the vascular and perivascular channels to cause abscess. It is in this latter way, in all probability, that ancient trauma is operative to cause brain abscess. It has been proved beyond question that such trauma may antedate abscess manifestations for many years. The varieties of injury most likely to cause brain abscess are those in which the injured tissues are lacerated by something which carries the infective process with it; thus penetrating wounds of the orbital cavities and punctured wounds of the epicranium which also penetrate the bone, or punctured fracture of the cranium itself, are attended with greater liability to this complication than are wounds produced by the surgeon.

The cases of brain abscess that occur from metastasis probably make up about one-fourth of the whole number. As has been said, metastasis is so common from the thoracic organs that this form has been referred to by some writers, for instance Martius, as pulmonary brain abscesses. It has been contended by Nather that the brain is the exclusive seat of metastatic formation from purulent lungs, and that the metastatic foci which result are always multiple. This, however, has been conclusively disproven by the investigations of Martius, who found that, in twenty-two cases in which disease of the brain occurred secondary to pulmonary disease, in six there was metastasis in other organs. In nine instances solitary abscess was found. Rarely, if ever, does brain abscess result from metastasis from other organs of the body, such as the intestines or liver. That it may occur from wounds of the extremities without the mediation of general sepsis is shown by a case reported by Turner, in which the disease was secondary to an injury of the arm received some weeks before. The diseases with which it has been demonstrated that brain abscess is associated most frequently are purulent bronchitis and bronchiectasis, gangrene of the lungs, purulent pleuritis, and pericarditis. Rarely has it been known to follow infective fractures of the bones of the extremities, periostitis, etc. In some of the so-called pulmonary brain abscesses lung pigment has been found in the brain (Böttcher).

The cases associated with aphthæ in which the oidium albicans was found in great numbers in the contents of abscesses have already been mentioned. A certain number of cases of abscess of the brain occur in which the causes so far enumerated cannot be made out. It is to these abscesses that the name idiopathic has been given. This class is constantly growing less, as it has been demonstrated that several forms of infection formerly considered incapable of producing purulent processes are shown to be possessed of this power, and practically idiopathic brain abscess exists only in the medical literature of the past. Among the infections that have been followed by brain abscess may be mentioned the tubercle bacillus, the bacillus of influenza, the coccus of erysipelas, and the diplococcus intracellularis of Weichselbaum. There still remain, however, after all these, a few cases in which no cause can be found.

The location of brain abscess has been proven to stand in rather close relationship to the origin of the causative factors. In a general way it may be reiterated that a statistical study of this disease shows the right side of the brain is more frequently affected than the left, although Pitt demurs to this opinion. Abscesses due to disease of the middle ear are almost always situated on the same side as is the purulent process from which they originated, and in a great proportion of cases either in the temporosphenoidal lobes or in the cerebellum, about three times more frequently in the former than in the latter. In very young children the abscess is very rarely in the cerebellum. This predilection of the temporosphenoidal lobe and cerebellum to abscess resulting from purulent middle-ear disease is dependent upon a number of causes, the most prominent of which, however, are the thinness of the tympanic roof and the greater liability to implication of the dura over the anterior surface of the bone than that over the posterior wall of the middle ear. It will be seen later on that the dura and brain substance between the disease and the abscess are frequently themselves the seat of the disease. In those cases in which there is healthy brain tissue between the temporosphenoidal abscess and the bone it is probable that infection has taken place or takes place either through the veins that enter into the superior petrosal sinus or through the perivascular lymphatics. And it is through the medium of the latter, the sinuses and lymphatics, that cerebellar abscesses occur. Of course brain abscesses of this origin are sometimes found in other parts of the cerebrum such as the occipital lobe, frontal lobe, pons, and cerebral peduncles, but compared with the frequency of occurrence in the portions of brain mentioned they are really very insignificant. In the cases of abscess from ear disease in which the dura and brain substance between the upper bony covering of the tym-

panic membrane and the abscess are not diseased, there will be found in some cases a thrombosis of the veins and sinuses which establishes a direct communication between the *locus* of original affection and the abscess, or there may be found a purulent infiltration following the course of the seventh pair of nerves. The acute infectious and exanthematous diseases cause brain abscess more by setting up otitis media purulenta than they do directly. Meslay has mentioned a case in which abscess of the brain was apparently directly due to the infection of an angina from which the child was suffering.

Abscess of the brain ganglia, the pons, and oblongata may occur from any of the general etiological factors. They are of rare occurrence, and generally due to pyæmia.

Abscess due to trauma may occur on the side of the brain which has been the seat of the original injury or it may occur on the opposite side; indeed, the latter is quite as frequent as the former. No such close connection exists between certain parts of the brain and the liability to abscess therein produced by trauma, as has been stated to exist from ear disease. In fact, all that can safely be said regarding the location of traumatic brain abscesses is that they are apt to be more deeply situated and as liable to be found in the poles or superior surface of the brain as at the sides. In metastatic brain abscesses there is a predominant tendency to occur in the distribution of the artery of Sylvius of the left side. These abscesses are metastatic and of embolic origin, and they occur on the left side of the brain for the same reason that embolic blocking up of brain blood-vessels does. For the same reason, likewise, these abscesses are more liable to be found in the central ganglia and midbrain than are abscesses of other origin.

No definite rule has been established as to the prevailing location of abscesses due to carious processes in the sphenoid and ethmoid bones, or of those resulting from purulent inflammation from adjacent cavities. The same may be said of the cases of abscess that occur apparently as true complications of infectious diseases, such as the cases reported by Bristowe.

Abscess of the brain is generally single and varies from the size of the end of a finger to that of a fist. They are not infrequently multiple, of very different size, the spots of suppuration having no connection one with the other. Von Bergmann has reported a case in which more than a hundred foci of suppuration were present in a case secondary to general pyæmia.

It will depend entirely upon the rapidity with which the purulent process develops and the duration of the disease whether or not the abscess will have a limiting membrane, that is, be encapsulated. If the

process has developed rapidly and is of moderately short duration, there will be no surrounding abscess wall. The process of inflammation will taper off from the centre of the abscess cavity where the tissue is entirely purulent towards the periphery through various grades of inflammatory reaction. Such abscesses are common in the cortical gray matter and are often associated with diffuse purulent meningitis. In cases that have been of moderately slow development and considerable duration, the purulent parts will be surrounded with a capsule which is more or less difficult to penetrate. It is no doubt due occasionally to these small abscesses that the symptoms of brain abscess, which sometimes come on after middle-ear disease or trauma, and which remain dormant for years, are lighted up and produce symptoms which are difficult to associate with the original injury. If, however, an abscess which has been thus encapsulated gradually has its surrounding barriers removed either by pressure or attrition and the contents of its cavity rupture into the lateral or other ventricles or on to the brain surface, there may result symptoms of acute abscess which are really due to a chronic process.

One of the most important recent discoveries of bacteriology is that pus becomes sterile in every old-standing collection which has no communication with the external world. In such encapsulated collections the microbes first lose their virulency, and although still able to grow on inert media, they have lost the power to overcome the resistance offered by living tissue or organism. Finally they lose their vitality, and eventually they become completely disintegrated. When such a condition occurs, however, the danger of a reinfection is always present to some degree, as such sterile pus forms a good medium for the growth and activity of new pyogenic organisms.

The fact that the limiting membrane which encapsulates the brain abscess is oftentimes sufficient to bring the activity of such purulent collections to an end, is shown by the records of cases in which single or multiple abscesses, firmly encapsulated, are found after death from other diseases.

The color of the pus, the odor, the specific gravity, and other physical characteristics depend in these cases, as they do in all other forms of purulent formation, upon the causative factors. When the destructive process has been a very rapid one the color of the pus is a reddish-brown. This is due to the presence of a considerable amount of disintegrated tissue. In some cases the disagreeable odor of the pus is very pronounced, and in these cases if the pus is examined the bacillus pyogenes fœtidus will generally be found. When the pus is of a greenish-yellow hue, examination will show the presence of the bacillus pyocyaneus.

Symptoms.

The symptoms of brain abscess are, in a small proportion of the cases (five to ten per cent.), as conspicuous by their absence as by their presence; that is, a number of cases has been reported in which practically no symptoms whatever were to be determined. For instance, Turner has reported a case of sudden death in a soldier who had been considered perfectly well. At the time of death he was reading a newspaper. On autopsy multiple abscesses of the left frontal lobe were found. No more convincing statement of the absence of definite pathognomonic symptoms in brain abscess can be made than a reference to the fact that of the seventeen cases analyzed by Martius in none was the diagnosis made or even suspected. As a rule, however, the symptoms may be classified into those depending upon infection or inflammation within the brain substance, those dependent upon the location, those dependent upon intracranial pressure, and the terminal phenomena. The symptoms in the beginning may depend somewhat upon the cause or origin of the abscess. For instance, occasionally in abscess due to purulent inflammation of the middle ear, the first symptom may be cessation of discharge, accompanied with pain in the side of the head, radiating from the ear. This may be continuous and violent or intermittent and paroxysmal. The same may be said of those due to trauma.

Frequently the beginning symptom will be thus directly attributable to the injury. In the majority of cases, however, unless the trauma be a penetrating one, such as the entrance of a nail or splinter of some foreign tissue into the brain, the original symptoms of the injury will have subsided before the symptoms of intracranial suppurative inflammation show themselves.

If after trauma, otitis media, or purulent inflammations of the pulmonary organs such symptoms of intracranial pressure and pus formation as somnolence, slowness of the heart's action, vomiting, restlessness alternating with stupidity, pupillary inequality, complaint of diminished vision or of diminished hearing, accompanied with rigors and slight elevation of temperature which, however, pursues no regular course, develop, it is a warrantable suspicion that purulent inflammation is going on within the brain. The most pronounced and constant symptoms are headache, generally of great and increasing severity, which is entirely unamenable to all forms of therapy, bradycardia, vomiting, and rigors, with slight elevation of temperature. These symptoms constitute more or less in every case the syndrome of the early or inflammatory stage.

After this stage has lasted for a time, varying from one to three days, the patient passes into a state characterized principally by obscuration of the mental faculties. He no longer complains of the excruciating pain, although the headache still persists and he complains when he is questioned about it. The patient is generally quiet, and lies in a semi-wakeful condition, with a tendency to doze, very like one comfortably drowsy. The countenance is devoid of expression of suffering, and a general lassitude and lack of sufficient energy to ask or answer questions attend this stage. The psychical reflex is slow, questions are answered in a delayed, monosyllabic manner, and it is next to impossible to prompt or rouse him to any connected thought or continuous statement. The similarity of this condition to that attending opium poisoning is very striking and has often been commented on. The pulse and temperature during this stage undergo alterations which are thought to be somewhat characteristic, in so much as they are relatively dissociated. The temperature keeps about normal, sometimes slightly elevated, more often a little below normal; but the pulse is slow, deliberate, full, oftentimes beating as slowly as 40 to 50 per minute. In the pulse the similarity to that found in opium poisoning is striking. Respiration is at this time altered in no characteristic way; generally it is somewhat slowed and occasionally there is a tendency to repeated sighing. The general symptoms are those of a beginning, profound asthenic condition. In addition to these, which are the most common, there may be, depending upon the location and multiplicity of the abscess, symptoms pointing to involvement of parts possessed of individualized function. For instance, if the abscess formation be in the temporosphenoidal lobe, there may be not only aphasia, which will be more fully described presently, but also, and particularly if the brain substance between it and the adjacent tympanic cavity be diseased, disturbance of hearing associated with considerable auditory paræsthesia. Many cases in which aphasia was a prominent symptom have been reported, notably by Trockenbrod, Eisenlohr and Sich, Lanz, Oppenheim, Jansen, Campbell, *et al.* An extremely instructive example of left temporosphenoidal abscess in which there were right hemiplegia, lateral deviation of the eyes, and aphasia has recently been recorded by Hughlings Jackson. If the lesion be of the central lobes and close to the cortex, there will be, first, irritation symptoms corresponding to the function of the lobes involved, and later corresponding manifestations of paralysis. For example, purulent accumulation or destruction in the left arm and face centre would be manifest first by twitching and spasm in these parts, and later by paresis or paralysis. So long as convulsions of a cortical character appear in a paralyzed

limb it is rather positive proof that the corresponding centre is indirectly irritated from some adjoining neighborhood, and is not itself the seat of the purulent focus. A knowledge of this fact is often of the greatest importance in guiding the surgeon. If the lesion be of the occipital lobes—a rare occurrence in brain abscess—early optic neuritis, associate visual disturbance, such as homonymous hemianopsia, and some degree of sensory aphasia will be prominent. In the same way symptoms involving the basal ganglia, cerebral peduncles, pons, and oblongata will betray themselves in more or less significant manner by causing symptoms which may be referred to the destruction of individualized parts of these structures.

In young children general convulsions occur more frequently than in the adult. In both cases they are indicative of severe infection and extensive lesion. Paralysis such as hemiplegia rarely occurs early, but when it does it indicates with considerable certainty purulent inflammation of the basal ganglia in the vicinity of the internal capsule, as was the case in a patient recently under my observation: a child developed symptoms pointing to abscess of the brain following a chronic purulent inflammation of the middle ear; at about the end of the first week of its illness a complete right-sided hemiplegia occurred. At the autopsy a considerable area of purulent and broken-down tissue was found in the locality of the left putamen.

In a considerable percentage of the cases a slight degree of hemiplegia develops during the course of the disease, particularly if the pyramidal fibres going from the cortex of either hemisphere are encroached upon.

The temperature range in cases of brain abscess is a fertile topic for contradictory statement; according to the experience of some it is usually high, while others commonly find it subnormal. This discrepancy in statement is due to the fact that many writers do not distinguish with clearness between acute and chronic brain abscess. As a matter of fact, when the disease occurs in children the temperature in the beginning and perhaps throughout the course of the disease may be considerably elevated, 101° to 103° F. A corresponding amount of brain inflammation in the adult might be accompanied by an insignificant or transitory febrile rise. This is in keeping with the common experience that the thermal balance is disturbed with extreme ease in childhood. As a rule, the temperature in cases of acute brain abscess is in the beginning elevated from two to five degrees; after the inflammatory symptoms have subsided it is usually from one to two degrees below the normal. If the disease be associated with a septic state of the blood the temperature phenomena characteristic of that condition will prevail.

The symptoms in the beginning of the disease are frequently very irregular, obscure, and not at all suggestive. After a variable time, however, the brain irritation symptoms will give place to symptoms of pressure and destruction. Psychical disturbance, manifested by restlessness, excitement, possibly delirium and disordered sleep, will give way to mental apathy, hebetude, sluggishness of mental processes, a condition which eventually terminates in complete coma. The pulse becomes slower, from 40 to 60 per minute; the force of its beat is irregular and occasionally its rhythm is profoundly disturbed. Respiration which in the beginning may have been accelerated and later slowed now assumes the Cheyne-Stokes type; vomiting ceases and is succeeded by difficulty of swallowing, more or less complete inability to take food. The nutrition of the patient alters with great rapidity, and day by day gradual dissolution becomes more manifest. The sphincters in this condition are relaxed. The surface of the skin, which at first was dry and hot, now becomes clammy, and the circulation, particularly of the extremities, is very depraved.

Information to be derived from examination of the patient in cases of brain abscess, particularly of the acute form, is frequently of great importance. Optic neuritis occurs in at least forty per cent. of all cases. The pupils in the beginning may be slightly contracted. After the disease has lasted sufficiently long for pressure symptoms to become manifest, the pupils will always be found dilated, less often irregular. The pupillary reflex to light and that in accommodation are preserved; the former, however, is usually sluggish; ocular paralysis is uncommon. Facial paralysis, disorder of speech, hemiplegia, hemiparesis, dysphasia, are symptoms that are sometimes found. In large temporosphenoidal abscesses where the pressure is great, adjacent parts of the disease focus which may be possessed of specific function may be pressed upon, and their function deranged. Pressure upon the lower portion of the ascending frontal and ascending parietal convolutions is accompanied by motor paresis of the opposite side of the body. Pressure upon the third frontal convolution, the island of Reil, and the adjacent ascending frontal convolution will be accompanied with more or less motor aphasia, while pressure on the posterior half of the first temporal convolution will be manifest by sensory aphasia, word deafness. Lesion of the third nerve, causing paresis or paralysis of all the eye muscles except the superior oblique and external rectus, may result from a large abscess in the temporosphenoidal lobes. If the seventh nerve be involved the resulting paralysis must be distinguished from that due to peripheral involvement in the usual way by the presence of

power to close the eye, by the possession of some emotional display on the paralyzed side of the face, and by the intactness of the sense of taste in the anterior two-thirds of the tongue.

In acute brain abscess examination of the reflexes shows no particular change, except possibly a more or less constant diminution of the cutaneous reflexes.

The two most important symptoms, then, in acute brain abscess are headache and increasing apathy. It would be fortunate if it could be held that there was anything characteristic about the headache of brain abscess; but there is not. It may be said, however, that it is persistent; that it is severer during the early night than in the early day; that it is increased by coughing, jarring, and stooping; entirely unamenable to any medication, and is generally accompanied by corresponding and increasing mental obscuration. It is not infrequently localized, particularly when the brain abscess is of otitic origin. But many cases have been reported in which pain in a remote area was dependent upon abscesses of the cerebellum on the opposite side of the head. No more are somnolency, apathy, and loss of mental ability characteristic of brain abscess. In fact, it must be stated that the previous possession by the patient of one of the three important etiological factors, followed by the symptoms which have been already detailed, with more or less localizing symptoms in individual cases, are the important findings that prompt us to a diagnosis. In some few cases it has been said that examination of the skull has facilitated the diagnosis. In a few cases there has been tenderness behind the ear, over the mastoid process. This condition is oftener associated with inflammation of the lateral sinuses than with brain abscess. In other cases percussion of the skull has been thought to be of some service. Lately, Macewen has stated that in his experience auscultatory percussion facilitates the diagnosis of intracranial changes. If the stethoscope be placed over the diseased area and that part of the skull be subject to percussion, a note of higher pitch and of greater intensity will be heard than over normal areas of the brain.

Abscess formation is so infrequent at the base, that basal irritation symptoms, particularly those referable to the individual cranial nerves, are very uncommon. If in addition to the severe cephalalgia and somnolency which have just been spoken of, we find slow pulse, normal or subnormal temperature, vertigo, disturbance of equilibrium, hebetude, maintenance of the prone posture, inability to raise the head, and desire to be left alone, it may be assumed that the most probable diagnosis is that of brain abscess. Localizing symptoms, which it is unnecessary to mention again, will further the diagnosis.

It has been stated that the second most common location of brain

abscess is within the cerebellum. In such cases very often there are in addition to the general symptoms of brain abscess symptoms which suggest the locality, such as early optic neuritis, which occurs in from sixty to eighty per cent. of the cases, persistent vertigo and vomiting, localized occipital headache, nystagmus, and pronounced difficulty of equilibration. In some cases, however, in which the cerebellum reveals an abscess cavity at the autopsy, there is an absence of all so-called cerebellar symptoms during life. If, however, there is with the abscess development a meningitis of the cerebellar fossa, there will be irritation symptoms, such as spasticity of the muscles of the neck and of mastication, profound depression, and pain radiating to the neck and between the shoulders, which will suggest the diagnosis. The cerebellum evidently betrays tumor involvement by more or less characteristic symptoms much more promptly and certainly than it does those of abscess. Cerebellar abscess is caused almost exclusively by purulent otitis, and abscesses of such origin are almost always single. There is generally a direct local extension of the inflammation from the posterior surface of the petrous bone to contiguous parts of the cerebellar hemisphere. In some cases, however, there is found a layer of apparently healthy tissue between the dura and the abscess.

Deafness has been mentioned by many writers as a differentiating symptom between cerebral and cerebellar abscess. It is of no great significance, however, as complete deafness is caused only by destruction of the labyrinth or affection of the eighth nerve, conditions that do not occur in cerebellar abscess.

The terminal symptoms of acute brain abscess usually occur as a gradual evolution of the disease process, and they correspond to involvement of new areas or to increased œdema of the surrounding tissue coexistent with the abscess.

If during the course of the disease the symptoms suddenly become of great severity, particularly if they assume a grave and prostrating type, associated with widely dilated pupils, marked disturbance of the pulse rate, abrupt elevation of temperature, and rapidly developing Cheyne-Stokes respiration, or if a paralysis of some individual nerve or member of the body quickly occurs, also associated with profound mental disturbance, it may be assumed that the abscess cavity has ruptured: in the first case into the lateral ventricles, in the second case on the surface of the brain. Cases have been reported in which a more or less profuse discharge of pus from the nose and mouth have been considered significant of discharge of brain abscess from the base of the skull. I am unwilling to admit such cases to the category of brain abscess, as they are more easily and rationally

explained by positing a purulent basal meningitis as the origin of the pus. That, however, the pus may escape through the softened membranes and the eroded tegmentum tympani is shown by cases recorded by Macewen. The latter observer has also seen a cerebellar abscess in which a sinus led through a carious erosion of the sigmoid groove into the abscess cavity which it thus drained. It is, however, so rare that a brain abscess cavity empties itself in this way that it is deserving merely of mention.

The symptomatology of chronic brain abscess requires a few words by itself. If the diagnosis of acute brain abscess is a difficult one, the problem involved in the diagnosis of chronic brain abscess is nearly insoluble. In the latter case the relationship between it and the productive factor or factors is much less obvious than in the acute form, and the injury on which it is dependent may have been forgotten. The otitis media purulenta may have been so slight, or of such little consequence, that the patient has neglected it, or the association between the development of brain abscess and some injury in a distant part of the body has been such an implausible one that it has not been taken into consideration. These forms of abscess may develop without causing any symptoms for which the patient seeks medical advice. They are almost always deeply situated and covered with a stratum of nerve substance which divides them from the meninges and cortex of the brain. Therefore symptoms which would bespeak their origin, as well as localizing symptoms are generally absent. The patient or those around him notice that he is more irritable, less tractable, and prone to inactivity; that his mental and physical vigor is constantly growing less, and that he complains of headache which is variable as regards both intensity and duration. In some cases a history of recurring convulsions will be given, and these may lead to a suspicion of epilepsy, while in others occurrence of forced, *manège* movements and a peculiar irritable, impressionable state may suggest hysteria. A case of the latter kind has been reported by Dana, in which the autopsy showed an old abscess in the right temporal convolutions. A similar case reported by Turner, in which there were absolutely no symptoms, has already been mentioned. A great many other cases might be cited, but a recent one by Kucharzewsky is instructive. In this case an abscess the size of a hen's egg was found in the right temporal lobe, secondary to a purulent otitis; the patient had complained of absolutely nothing except severe headache. There were no focal symptoms whatever; three days before death he suddenly became unconscious.

It should not be inferred that focal symptoms and symptoms of intracranial pressure may not be present in chronic abscess.

They may be present in a case in the beginning of the disease and then completely subside, and so symptomless may this period of latency be that the patient will continue at his labors or pleasures for an indefinite time until some new injury to the head or infection from a distance starts the latent into an active stage; then death follows within a few days. When the acute stage of chronic abscess does occur, the patient usually succumbs very rapidly, either with purely asthenic symptoms, dependent upon intracranial pressure or rupture symptoms, ventricular or meningeal.

In all these cases of chronic abscess the temperature is as a rule below normal. Murri states "that a subnormal temperature of long duration and not pyrexia is an accurate sign of chronic abscess of the brain."

Diagnosis and Differential Diagnosis.

The diagnosis of acute abscess of the brain is always a difficult one, because there are no absolutely pathognomonic symptoms. The clinical picture to which it gives rise may be closely simulated by acute encephalitis, by purulent disease of the lateral sinus, by purulent leptomeningitis, particularly of traumatic origin, very rarely by peripetrous abscess and septic process in the mastoid cells. Other diseases, which it would seem on first thought have little in common with the symptoms of brain abscess, may also be confounded with it. Brief reference to a patient recently under my observation will show this:

A man, previously in excellent health, fell from a second-story window while intoxicated, and struck on the head. A large, open scalp wound, associated with symptoms of concussion of the brain, were the immediate results. At the end of a fortnight the scalp wound had healed, but the patient complained of dizziness, constant headache, loss of appetite, etc. He was apathetic, non-communicative, and in a dream-like state of consciousness. The temperature was almost continuously elevated, ranging from 99° to 103° F., sometimes higher in the morning than in the evening, or *vice versa*. Added to these, there were the usual septic febrile phenomena. The pupils were continually dilated, and there was profound and progressive muscular weakness. On account of the trauma and the apparent genesis of the symptoms therefrom, abscess of the brain was strongly suspected and the patient was carefully watched. Although there had been no symptoms, such as cough, pain, or shortness of breath, to prompt a suggestion of pulmonary trouble, routine examination of the chest revealed double apical infiltration, which increased from day to day. Microscopical examination of sputa made later showed the presence of tubercle bacilli. The disease ran an un-

interrupted course. The previous trauma, followed by a period of latency, then febrile symptoms of a septic character, associated with headache, vertigo, lethargy, dilated pupils, and progressive emaciation, were strongly suggestive of brain abscess, and the suspicion could not have been eliminated except by the detection of some other disease.

The recital of another case will show the difficulty of diagnosing brain abscess from purulent meningitis:

A man, of about 40, had the ferrule of an umbrella handle jabbed into his eye during an altercation in the street. He was taken to the hospital, and after some days the eyeball was enucleated on account of beginning cyclitis. At this time there were symptoms suggestive of delirium tremens; these subsided, and at the end of a month the patient was mending and progressing satisfactorily. About this time he began to have headache, elevation of temperature, periods of sleeplessness, alternating with stupor; later there was slight stumbling in speech and incoherency, and still later defective innervation in the area of the right fifth nerve and gradually increasing apathy, Cheyne-Stokes respiration, and the usual accompaniments of the profoundly asthenic state. Much care was taken in the endeavor to substantiate the diagnosis of brain abscess, and had not the case been one in which criminal procedure was anticipated and the consent of the family was withheld, an exploratory trepanation would have been made. At the autopsy the lesion proved to be a purulent leptomeningitis of the lateral and basal portions of the hemispheres. On opening the hemispheres and lateral ventricles, it was seen that the tela choroidea and choroid plexus were laden with purulent accumulation. No foci of purulent destruction were found in the brain substance.

This case illustrates the absolute impossibility of always differentiating brain abscess from purulent meningitis. The diagnosis of meningitis following on trauma, as well as meningeal hemorrhage and cortical inflammation and hemorrhage, will be prompted by the relatively short period of time between the trauma and the development of symptoms, by the development of such irritation symptoms as stiffness of the neck, by the more common involvement of some of the cranial nerves, and by the absence of focal symptoms. In cortical inflammation and hemorrhage of traumatic origin the symptoms will come on early, but in neither case will there be any indications of suppuration. If hemorrhage be of considerable extent, it will, depending upon its location, produce focal symptoms, as well as symptoms of compression. These symptoms will come on soon after the injury and be directly traceable thereto. It is when these pathological conditions take on a purulent infection, which they sometimes do, particularly if the disease be prolonged, that the diagnosis is attended with great difficulty. It is quite impossible to make an early differ-

entiation between acute encephalitis, particularly if it be preceded by trauma, and beginning brain abscess. The latter is always accompanied by the symptoms of acute encephalitis in the beginning, while the former, if it be dependent on infection, goes on to suppuration, so that it is necessary to wait for a time before the diagnosis can be made. The occurrence of any of the phenomena of sepsis, such as rigors, vacillating temperature, etc., are very important indicative factors of abscess.

The disease with which chronic brain abscess is most liable to be confounded is brain tumor. As has before been stated, the development of chronic brain abscess may be long delayed after the occurrence of etiological factors on which it is dependent. The course of the disease is itself very slow and misleading. Frequently the symptoms are none of them of sufficient severity to call for particular notice until after the disease has existed for a long time. It may be said, however, that the history of any septic condition, the presence of a purulent focus in any other part of the body, together with the occurrence of rigors, slight elevation alternating with rather constant depression of bodily temperature, a slow pulse, headache varying in severity and increased by anything that increases brain pressure, manifestations of psychical perversion in the shape of hysterical symptoms or hypochondria—all speak in favor of chronic cerebral abscess more than they do of brain tumor. Further, it may be said that optic neuritis and focal symptoms are much more apt to be present early with brain tumor than with chronic brain abscess. This is so particularly because the latter has a special predilection for one portion of the brain, namely, the temporosphenoidal lobes, and there is a marked tendency in all of these slowly developing abscesses to become firmly encapsulated. When acute manifestations are superimposed on those of chronic abscess, there is not so much necessity to differentiate it from tumor, as both conditions—chronic brain abscess and brain tumor—require the same therapeutic measures.

The failure to make a differential diagnosis is not such an important one, providing a diagnosis be made. Of course, in the latter condition a central situation may preclude the presence of sufficient symptoms to make a diagnosis, while on account of the ease with which an abscess cavity may rupture into one of the lateral ventricles, the patient's life is more imminently jeopardized than in brain tumor. It is then to the origin, to the prolonged period of latency, to the slight manifestations of suppuration, and to the supervention of acute symptoms upon the chronic, that we must look for the differentiation of the two conditions.

Oppenheim has pointed out the necessity of making a differential diagnosis between brain abscess and the traumatic neuroses. Unquestionably, these conditions may sometimes be confounded, particularly as hysterical and hypochondriacal symptoms are occasionally present with abscess of the brain. The diagnosis between these two conditions can only be arrived at by the most careful daily observation. The variability of symptoms in the latter condition, or their persistency without manifestations of bodily or mental harm to the patient, will prompt the diagnosis; and absence of optic neuritis, persistent severe headache, and manifestations of suppuration will confirm it. A diseases with which brain abscess is most liable to be confounded is a state with which it may be associated, namely, thrombosis of the lateral sinus, and particularly purulent thrombosis. This condition not infrequently develops with disease of the middle ear and of the mastoid cells. The most weighty factor in its diagnosis is tenderness, swelling, and œdema over the mastoid process on the affected side, and dulness on percussion. These, associated with other evidence of unilaterality of the symptoms, are the weightiest factors in leading to a diagnosis. In purulent disease of the lateral sinus, the temperature is very much higher than in brain abscess; and the pulse, unlike that of brain abscess, is rapid, weak, and keeps pace with the temperature. Optic neuritis and focal brain symptoms are naturally absent with disease of the lateral sinus.

General pyæmic symptoms, such as attacks of colliquative sweats and diarrhœa, evidences of infarction in the lungs, and tense, overfilled, rigid jugular veins on the side corresponding to the thrombosis in the sinus, will corroborate the diagnosis.

Prognosis.

Abscess of the brain is a condition which seriously jeopardizes life in every instance. Time was, and that not long ago, when its occurrence was uniformly fatal; but now, when operative surgery is such a willing and reliable handmaid of the physician, this disastrous outcome occurs in but a comparatively small part of the cases that are diagnosed early.

It must be said that the prognosis is even now a very serious one. Every case not diagnosed and subject to operation terminates, sooner or later, in death. It has been contended that the contents of abscess cavities might undergo retrograde changes which would promote their disappearance or absorption, and that the abscess walls, then falling together, might occlude the former cavity. This belief does not conform with the tenets of modern knowledge of the laws of

repair. It is unquestionable that sufficiently well-marked symptoms of brain abscess do occur to justify a diagnosis, and that these symptoms may subside and disappear. In these cases, however, it is well to remember that the purulent focus is still in the brain tissue, although the pus is sterile and encapsulated, and may at any time be stirred into active existence by the presence of some malign influence, such as infection or trauma.

The prognosis of brain abscess depends, in part, upon the causation. Its occurrence is attended with greater prospects of recovery when dependent upon ear disease. And this for two reasons. In the first place, the previous presence of purulent discharge from the ear is a weighty element in leading to a diagnosis; and secondly, it can be stated, with a fair degree of positiveness, that localization of otitic brain abscesses is more certain than that of those due to other causes, and, therefore, there is greater prospect of finding them on operation. The prognosis is most serious when the disease is due to metastasis. In the first place, the original source of the emboli is usually in itself a very serious disease, and when the prostrating accompaniments of brain abscess are added to it, the patient usually succumbs.

When the abscess is a slowly developing one, it is, as has been stated, liable to be surrounded with a well-developed protective capsule, which may be the means of prolonging the patient's life sufficiently to make the localizing diagnosis and its removal possible, providing the abscess is in an accessible locality; or changes may go on in the abscess wall and abscess contents which render the presence of the foreign mass not strictly inimical to life. This has been shown by cases in which death has been due to other diseases, in which ancient suppurative foci in the brain have been found at the autopsy.

In short, it may be said that the most weighty question bearing on prognosis is, Is the abscess diagnosable and localizable? If it is, the prognosis is for recovery in about two-thirds of the cases. If it is not diagnosable and localizable until after the patient has been exhausted by long-continued suffering and the presence of an infected suppurating mass, the prognosis is most serious. If no diagnosis is possible the disease will tend unerringly to a fatal termination.

Treatment.

The preventive treatment is that towards which the physician should turn his attention. If it be kept in mind that from one third to one-half of all cases of abscess of the brain are due to a disease, otitis media purulenta, which is ordinarily not looked upon as

one dangerous to life, and which we believe to be extremely amenable to treatment, it will be readily seen that this condition is one which demands the urgent and careful attention of the physician. It is out of place in this connection to enter into a discussion of what these measures are. The other etiological factors are not so easily obviated, although much may be done by keeping wounds of the scalp and cranium in an aseptic condition. Wounds of the dura should be freely opened and treated antiseptically. No measures will prevent metastasis from a gangrenous lung, nor does treatment seem to rob influenza and the specific diseases of their malign tendency; but it may be assumed that measures that contribute to improve the bodily vitality tend to prevent all complications, brain abscess among the rest. The same may be said of maintenance of the circulation in children with patency of the foramen ovale; "blue babies" are very liable to brain abscess.

When abscess occurs, there is but one form of treatment to be thought of, and that is operative. The physician should urgently advise operation in every case of brain abscess in which a diagnosis is made with a reasonable degree of certainty. It matters not whether a diagnosis of the exact location of the abscess can be made, the necessity of entering that skull cavity in search of the suppurating mass remains quite as urgent. I am aware that v. Bergmann, past master of the surgical art in Germany, has said that the cranium should not be opened unless the surgeon is certain that his instrument will find the lesion of the brain in the exact spot and under exact conditions foreseen previous to the operation. Such teachings are pernicious but I trust not far-reaching. In cases in which focal symptoms indicate certain areal involvement, the earlier that portion of the brain is laid open the greater will be the chances of recovery; for if the physician procrastinates on account of an indefinite feeling that the diagnosis is ill-founded, or that something miraculous will happen to clear up the symptoms, the disease may have time to make such an onslaught on the vital powers of the patient, both by increasing intracranial pressure and by causing destruction of essential parts of the brain, that shock, which attends all brain operations, will be enough to sever the sufferer's grip on life.

Considering the frequency with which two localities of the brain, the temporosphenoidal and cerebellar lobes, are the seat of abscess, it would seem the rational plan of procedure, when the symptoms of brain abscess are lacking in localizing accompaniments, to open the skull over one or both of these areas, electing for the first operation the site indicated, indefinitely perhaps, by certain symptoms. For instance, if to the usual complex of symptoms going to make up the

history of a patient with brain abscess there are optic neuritis and persistent vertigo, the involvement of the cerebellum would be more probable than that of the temporosphenoidal lobe, although the latter might be the seat of suppuration and cause these two symptoms in addition to the rest. If, after opening the skull and exploration with the probe, no pus has been found, very little danger to the patient's life has been contributed, and one that every one would risk if in condition to give an opinion, considering the absolutely fatal outcome when this disease is left to itself. Of course, the hesitancy in resorting to surgical procedure will come through uncertainty of diagnosis; then the question to decide is whether exploration is not justifiable to facilitate the diagnosis. I am of the opinion that it is, providing other conditions simulating the symptom complex of brain abscess can be excluded. The extremely favorable outcome of the many cases of brain abscess in which early, or even delayed, surgical interference has been resorted to, and the comparative innocuousness of opening the skull cavity, leads me to this conclusion. Brain surgery has not fulfilled the promise which it a few years ago held out in any intracranial disease, except in sinus thrombosis and the disease under consideration; but here it has more than equalled even sanguine expectations. The results obtained by Macewen, by Dean, by Winter and Deanesly, and others, by operation for cerebellar abscess, should encourage surgeons not to delay the application of the only means that can save the patient's life. All operative procedures, such as opening the mastoid and the like, unless there are special indications for them, are to be deprecated, and the operation that should be done is trepanning of a sufficiently large opening in the skull to allow of proper and complete exploration, to evacuate the abscess cavity, and to admit of and facilitate free drainage. If, on account of the absence of focal symptoms, the point for election of operation is not indicated, then the general rules, as given by Ballance for opening the skull and illustrated by the accompanying figure, should be followed. The temporosphenoidal lobe is best reached through a trephine opening, one and three-fourths inches above, and one and one-fourth inches behind the external auditory meatus. An opening of this large size should be made not only to facilitate possibly necessary exploration. but because it is the experience of surgeons that a large trephine opening in no way adds to the danger of the operation and is less liable to be followed by hernia of the brain and infection of the meninges.

The process that goes on in the abscess cavity after the evacuation of pus is very similar to that in abscess in any other part of the body. In acute abscess, in which the process of development has been very rapid, even before the semblance of a limiting membrane had formed,

FIG. 12.—Lateral Aspect of a Small Adult Skull (Ballance). The illustration shows the relations of the lateral sinus to the outer wall of the cranial cavity and the position of the trephine opening (*a*) which should be made when it is deemed necessary to expose it. The base line (Reid's) passes through the middle of the external auditory meatus and touches the lower margin of the orbit; it is marked out in eighths of an inch, as are also the perpendicular lines drawn from it. The measurements are made along the base line from the middle of the bony meatus. The drawing also shows the convolutions of the temporosphenoidal lobe, the Sylvian fissure, and the position of the lower end of the furrow of Rolando (Rol.). *xx* Indicates the site of the tentorium as far as it is in relation to the external boundary of the skull. The anterior *x* shows the point where the tentorium leaves the side of the skull and is attached to the superior border of the petrous bone. *a*, Trephine opening to expose sinus, five-eighths of an inch in diameter, its centre being one inch behind and a quarter of an inch above the middle of the bony meatus. This opening can easily be enlarged upwards and backwards and downwards and forwards (see the dotted lines) by suitable angular cutting bone forceps. It is always well to extend it forwards, so as to open up the mastoid antrum (*c*) and the gutter of the carious bone (if there be one) which leads from the antrum, tympanum, or meatus down to the bony groove. The position of the trephine openings which must be made for the relief of inflammatory intracranial affections secondary to disease of the ear other than for sinus pyæmia have been added to the drawing for the sake of contrast and completeness. They are as follows: *b*, Trephine opening to explore the anterior surface of the petrous bone, the roof of the tympanum, and the petrosquamous fissure, half an inch in diameter, its centre being situated a short inch (seven-eighths of an inch) vertically above the middle of the meatus. At the lower margin of this trephine hole a probe can be insinuated between the dura and bone and made to search the whole of the anterior surface of the petrous. *c*, Trephine opening for exposing the mastoid antrum, a quarter of an inch in diameter, and half an inch behind and a quarter of an inch above the centre of the meatus; or a quarter of an inch above the centre of the meatus and a quarter of an inch behind its posterior border. The trephine should be directed inwards and slightly downwards and forwards. When a superficial disc of bone has been removed it is well to repeat the operation with the gouge. A larger trephine may with advantage be employed, especially in adults. *d*, Trephine opening for temporosphenoidal abscess, half an inch in diameter. Situation recommended by Barker, one inch and a quarter behind and one inch and a quarter above centre of meatus. The needle of the aspirator is to be directed at first inwards and a little downwards and forwards. Birmingham prefers one and three-fourths of an inch above in order to avoid the lateral sinus. *e*, Trephine opening for cerebellar abscess half an inch in diameter and one inch and a half behind and a quarter of an inch below the centre of the meatus. Birmingham prefers two inches behind and one inch below to avoid the occipital artery. The anterior border of the trephine should be just under cover of the posterior border of the mastoid process. The drawing shows that a trephine hole made in this situation is far away from the lateral sinus, and that the trocar and cannula of the aspirator, if directed forwards, inwards, and upwards, would hit an abscess occupying the anterior part of the lateral lobe of the cerebellum, which is the usual site of collection of pus in this part of the brain.

escape of the pus is quickly followed by obliteration of the cavity merely by expansion of the brain about it. In slowly developing and in chronic abscesses increased intracranial tension does not facilitate emptying of the abscess cavity, and as the remaining pus and that gradually produced from the limiting membrane disappears through draining, the abscess walls fall together, and the gap heals by the formation of granulation tissue. As these granulations develop from the bottom, when the reparative process reaches the surface it unites and anchors the pia to the cortical cicatrix. Macewen has shown that the physical after-effect of this is akin in its physiological result to a blow applied to the cranium. It is apt to cause unconsciousness, generally of brief duration, which may, however, recur frequently and be accompanied by the development of a more or less extensive encephalitis. He thinks also that this cerebral irritation might lead to epileptic fits, although no instances following operation for cerebral abscess are recorded so far as I can determine.

Not infrequently convalescence after operation for brain abscess is a slow one, and measures contributing to bettering of the patient's general nutrition must be pushed vigorously.

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Superior Acute Polioencephalitis.

(Nuclear Ophthalmoplegia.)

This affection is a form of ophthalmoplegia dependent upon an acute hemorrhagic inflammation of the central gray matter in the floor of the third ventricle and the aqueduct of Sylvius, and analogous to the acute hemorrhagic form of encephalitis. It was first described by Wernicke in 1881. He based the description and the endeavor to establish the disease as a clinical entity on three cases in which after death, a termination quickly reached, acute hemorrhagic inflammatory destruction was found in the gray matter around the third ventricle.

For a number of years after Wernicke gave the description of these cases observations by other authors were very few, in fact limited to those of Gayet, which had been observed before Wernicke's, and of Thomsen. In the cases reported by these three authors it was pointed out that the disease seemed to have but one causation—alcoholism, and one termination—death. It may be remarked also that the cases were all reported by German observers. These facts would indicate that the disease is a very uncommon one. Latterly other cases have been reported which, although they do not bespeak the more frequent occurrence of the affection, show that the original claims in regard to the genesis and termination of the disease are without foundation. Reports of cases made by Uthoff, Salomonsohn, Wolfe, and others show that the disease may occur in those who are abstainers from alcohol, while the report of the latter, as well as those of Thomsen, of Werner, of Boedeker, and of Herrnheiser show that the disease may terminate in complete recovery. A recent writer, Jacobäus, would have us believe that the disease is a complication of alcoholic polyneuritis. Granting that this is not only possible but that it does sometimes occur, it is impossible to thus dispose of the causation of all cases.

It would now seem to be quite well established that next to chronic alcoholism the same acute infectious process that causes the form of acute hemorrhagic encephalitis previously described causes this form also, viz., influenza. It occurs also after pneumonia and diphtheria. These two diseases, therefore, differ only in the location of their pathological process and in their course and termination, not in their causation.

In addition to these it has been observed after sulphuric-acid poisoning, in poisoning by lead and by oxide of carbon. In one case the only attributable cause was a severe fright.

Symptoms.

Although the onset of the disease is usually unheralded, there may be such premonitory symptoms as occasional attacks of vertigo, peculiar sensations in the extremities, diplopia or other disturbance of vision, headache and uncertainty of gait—symptoms which indicate some form of intoxication. Or the first symptoms may be, and frequently are, in alcoholic patients, stupidity, somnolency, mental confusion, delirium; in fact, the mental symptoms may be those attending an attack of delirium tremens. Stiffness of the neck, vomiting, slight elevation of temperature, although the disease is a feverless one, may precede the paralytic symptoms for a short time, but

generally within three or four days some ptosis, fixed position of one or both eyeballs, or internal deviation due to paralysis of the external muscles of the eyes indicates the beginning of a progressive external ophthalmoplegia which within a short time becomes complete on both sides. The internal muscles are but slightly affected if at all, and the levator palpebræ muscle is unaffected. Associated with this there are generally symptoms of involvement of the second nerve, a varying degree of optic neuritis and corresponding disturbance of vision, more or less complete ptosis and ophthalmoplegia, although it must be mentioned that sometimes single muscles, such as the superior levator of the eyelid or the sphincter of the pupil, may be spared. In such cases there will be no change in the pupil and no ptosis. With the ophthalmoscope there can frequently be seen during the first few days of the disease numerous punctate hemorrhages and capillary extravasations around the papillæ, and congestion of the veins in the zone about the macula. When the ptosis is but partial there is still ability to close the eyes, but efforts to squeeze the eyelids tightly together are without result. Rarely have symptoms pointing to involvement of the seventh nerve been noticed. Yet diplegia facialis was a pronounced symptom in the case reported by Wolfe, and paresis of the seventh nerve on one side is recorded by Jacobäus.

Disturbance of gait varying from slight incoördination to one so reeling and uncertain as to suggest cerebellar ataxia, and also increasing weakness of the extremities with diminished or absent reflexes occur coincidently with, or before, the ophthalmoplegia. In one case this motor weakness was so pronounced on one side of the body as to constitute distinct hemiplegia (Gayet). In some cases there are disturbances of speech, particularly if there be any paralysis of the seventh nerve and of the extremities.

The partial or complete ptosis, the internal convergence or fixedness of the eyeballs, and the whimpering, pathetic, facial expression go to make a striking appearance. Usually the sensory sphere remains unaffected. As has been said, if the disease follows infection there may be in the beginning a variable degree of fever, but in the cases attending chronic alcoholism the temperature is often subnormal. The pulse is small, frequent, and gradually grows more feeble. The restlessness of the delirious, incoherent patient is superseded by a stuporous, comatose condition, in which he succumbs as a rule before the end of the second week. In the beginning, when the patient is in the stuporous, somnolent condition, he can be aroused with great difficulty, and possibly he may answer intelligently, but he immediately relapses into stupor. In the cases that have terminated in recovery the symptoms never reached the more profound stage, and the

progress of recovery was gradual after the symptoms had lasted from one to two weeks. An observation of Gayet shows that the disease may last several months; it is then properly referred to as subacute. It may, indeed, become chronic. This condition may be described as superior chronic polioencephalitis. Then the clinical picture is none other than that described by Hutchinson and Graefe as chronic progressive ophthalmoplegia. It should be stated that this observation is unique.

It has been remarked that the psychical manifestations in the cases due to alcohol are very similar to those so common to alcoholic polyneuritis. It is natural to infer that the cause of such psychical perversion is not the lesion of the gray matter around the third ventricle, but the complicating cortical changes of an unknown nature. To this latter must likewise be attributed anomalies of sensation, disturbances of the reflexes, and other inconstant manifestations.

Morbid Anatomy and Pathology.

On autopsy, cross section through the brain ganglia reveals the brain substance around the walls of the third ventricle for a variable distance to be of a dark rose-red color, of softened consistence, and the seat of numerous punctate hemorrhages and hemorrhagic extravasations. In some cases the inflammatory manifestations are not confined narrowly to the gray matter of the third ventricle. In one of Wernicke's cases small hemorrhages and hemorrhagic foci were found in the posterior quadrigeminal body, and in one of Thomsen's similar changes existed in the body of the corpora quadrigemina and in the posterior commissure. Microscopically the hemorrhages are seen to be confined principally to the vessel walls, with here and there distinct extravasations. The small blood-vessels and capillaries are distended and overfilled, and around them there is an increased number of granular and spindle cells. These changes are usually confined to the immediate environs of the third ventricle and may be traced into the gray matter of the aqueduct as far as the floor of the fourth ventricle. If there be more extensive involvement other symptoms indicative of destruction of nuclei situated in the diseased areas will be superadded. The blood-vessels in other parts of the brain have been found in an apparently healthy condition, but more often they are the seat of degeneration.

The pathology of this affection does not differ from that of acute hemorrhagic encephalitis.

Treatment.

The general measures to be adopted in the treatment are the same as for the form of encephalitis previously considered. It is only in the cases following influenza and other infectious processes that any hope of recovery can be held out. And in these it is not possible to say how much treatment contributes to that end. An insidious onset, subacute course, and the absence of subnormal temperature are symptoms that point to a favorable outcome; just as an abrupt onset, bizarre course, subnormal temperature, even without an alcoholic history (which always predicates dissolution), indicate a most grave prognosis.

Acute Polioencephalitis Inferior and Polioencephalomyelitis.

Before dismissing the subject of acute polioencephalitis it is necessary to devote a few paragraphs to the mention of a similar inflammatory process in other parts of the central nervous system. It has been said above that the pathological process in acute superior polioencephalitis does not always confine itself to the gray matter around the third ventricle; it may extend in the same substance beyond the aqueduct of Sylvius to the fourth ventricle, and even into the cord. The clinical picture will vary, depending upon the level of the lesion and upon involvement of the groups of ganglionic cells which are the nuclei of origin of the cranial nerves. We have seen that when the lesion is confined to the gray matter of the third ventricle, the third, fourth and sixth nerves are involved. The pathological condition resulting is known as polioencephalitis superior, which causes a progressive external ophthalmoplegia, whereas when the inflammatory process extends caudad to involve the nuclei of origin of the tenth and twelfth nerves the condition is called polioencephalitis inferior. These two conditions not infrequently exist together and the cases which have been reported show that they sometimes follow influenza. The clinical syndrome of the affection is that of superior polioencephalitis plus bulbar symptoms. It is only recently that these cases have been properly interpreted. When polioencephalitis superior and inferior have existed together the prognosis has been considered unequivocally bad, but reports of cases of recovery by Uthoff and Oppenheim and probably also one by Gayet have overthrown that dictum. When the disease is confined to the medulla oblongata it is known as poliomyelitis bulbi and it is considered briefly under that heading. When the inflammation constitutes not only polioencephalitis superior and

inferior but poliomyelitis as well, the name poliencephalomyelitis or polioencephalomyelitis is given to it. A much larger number of such cases are to be found in the literature than cases of polioencephalitis superior or poliomyelitis bulbi alone.

Thus we see that inflammation of the gray matter of the central nervous system may be localized in the cortical gray matter, acute hemorrhagic encephalitis; in the gray matter surrounding the third ventricle and the aqueduct, superior hemorrhagic polioencephalitis; in the gray matter surrounding the floor of the fourth ventricle, inferior hemorrhagic polioencephalitis; in the central gray matter of the spinal cord, poliomyelitis. Furthermore, the morbid anatomical changes found in all these conditions are very similar and they may be and often are immediate sequences of infectious disease. With one exception, superior polioencephalitis, the most common cause of which is alcohol, the etiology of these diseases is the same in every particular. They all occur most frequently in the young and in the wake of acute diseases; and the clinical course of the various conditions does not differ very materially. Cases of inferior polioencephalitis and polioencephalomyelitis are apt to run a somewhat more subacute course than those of superior polioencephalitis, but to this rule there are the most striking exceptions. Such a one has recently been reported by Dinkler. The case was that of a five-year-old child, who developed, after a fall on the head two and a half years previously, symptoms of motor unrest, deterioration of mental faculties, disturbance of speech, and attacks of headache and vomiting. Then suddenly there occurred without warning, high fever and coma followed by death within forty-eight hours. On examination of the central nervous system the typical lesions of inferior polioencephalitis, or better, polioencephalomyelitis, as the extravasation involved the posterior gray matter of the cord, were found. The extravasations were principally in the nuclei of the eight lower pairs of cranial nerves and in the main confined to the superficies of the floor of the fourth ventricle and central canal. The hemorrhagic conditions were very recent and severe and they had so quickly overwhelmed the vital resistance that the patient succumbed within a few hours.

Polioencephalitis superior and inferior and poliomyelitis anterior may occur simultaneously and be the single pathological expression of the same cause, and terminate in death. Such a case following influenza has been noted by Goldflam. Another case of similar symptoms but of unknown origin terminating in recovery has been recorded by the same observer.

In cases of inferior polioencephalitis the external muscles of the eye are first involved, and there soon follows paralysis of a

muscle or group of muscles of the extremities which bespeaks involvement of certain ganglionic cells in the anterior cornu. This paralysis in an extremity is followed by atrophy in a way characteristic of anterior poliomyelitis. The symptoms which indicate involvement of the spinal cord may precede those indicative of lesion in the oblongata, such as paresis of the tongue, of the seventh nerve, defective innervation, and diminution of the reflex irritability of the soft palate, difficulty of swallowing, and possibly attacks of dyspnœa, tachycardia, with elevation of temperature, symptoms which postulate affection of the nuclei of the seventh, tenth, eleventh, and twelfth cranial nerves, or the spinal symptoms may follow them.

Although recovery may occur in these cases (Goldflam), yet the prognosis must always be a doubtful one. That the appearance of bulbar symptoms in this subacute form is not the signal for an absolutely bad prognosis, as was contended by Guinon and Parmentier, is shown by more than one report of recovery after pronounced bulbar symptoms had existed for some time. As has been emphasized by Oppenheim, the symptoms of great gravity which predicate early dissolution are those indicative of involvement of the white matter.

The morbid changes in polioencephalitis inferior do not differ in nature from those of the superior form, with which they are often associated. Very often, in addition to the hemorrhagic inflammation and extravasations around the nuclei of the central gray matter, other structures which are not usually considered a part of the central gray matter are involved, such for instance as the nucleus of the facial, the substantia gelatinosa, the ascending root of the fifth, all of which were involved in a case studied by Kaiser. It is probable, in cases in which there is inflammatory affection of the white matter, that it is an expression of the severity and duration of the lesion, the same as affection of the white matter of the spinal cord in cases of acute poliomyelitis indicates profound and continued inflammation.

Whether or not a chronic form of encephalitis exists which we are incapable of recognizing microscopically or macroscopically it is as yet impossible to say. It suffices in this connection to remark that the clinical picture of polioencephalomyelitis has been seen in a number of cases in which no anatomical basis or foundation could be established after death, a termination which some of the cases is reached. This subject is considered at some length under the caption, Asthenic Bulbar Paralysis.

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Syphilitic Encephalitis.

When the term brain syphilis is used, it implies nearly always syphilitic disease of the membranes, of the blood-vessels, of the substance of the brain, or of the cranial nerves in their intracranial extent. Although frequently two or all of these conditions occur simultaneously and constitute one disease, it has been considered most fitting to treat these conditions in the sections devoted to each respective subject, instead of grouping them under the one title of syphilis of the nervous system. Syphilis of the brain, in the limited sense of the term, includes only gumma of the brain substance (which is considered in the article on Tumors of the Brain), and diffuse and circumscribed syphilitic encephalitis.

Diffuse syphilitic encephalitis consists really of the occurrence of diffuse gummatous formations in the gray and white substance of the cerebral hemispheres, particularly in the gray, more rarely in other parts of the brain, and entirely divorced from meningitis and meningo-

encephalitis. The starting-point of the syphilitic formation is the tela choroidea and the blood-vessels. The new formation consists of a diffuse round-cell infiltration, with proliferation of connective-tissue cells with new vascularization. This undergoes retrograde changes, hyaline degeneration of the vessels, and caseation of the mass similar to those of gumma. In the circumscribed form of syphilitic encephalitis, the newly formed masses of sclerosed tissue are distinctly limited and disseminated, like islets of multiple sclerosis, but differing from them, insomuch as they have a predilection for the surface of the hemispheres and a tendency for the mass to undergo fatty metamorphosis. Histologically, these little patches differ from the diffuse variety by the relative preponderance of connective tissue, by the comparative absence of blood-vessels, and by the hard, resistant feel. Naturally they produce destruction of the tissue areas in which they develop, as well as vascular and connective-tissue cell reaction. This gives the patches and their immediate environment a striking but not a characteristic appearance—grayish-white or yellow in the centre, and grayish-red towards the periphery. It is more than probable that in some instances these lesions have been described under disseminated insular sclerosis (which see). To quote one instance in support of this statement: Du Cazal reports a case in which such manifestations of brain syphilis as headache and attacks of epilepsy came on four months after infection. They yielded readily to antiluetic treatment. The time of occurrence of these lesions, computed from the time of luetic infection, is very much shorter than was formerly supposed. When the manifestations of syphilis were classified on a temporal basis into primary, secondary, and tertiary, it was customary to state that these encephalitic lesions, like other forms of brain syphilis, were late tertiary manifestations. Such a contention is entirely untenable, and is claimed at the present time by but very few. At least one-half of the cases occur within the first three years after infection. In the experience of Braus, forty-four per cent. of brain syphilis occurs within the first year; and Mauriac states that forty-three per cent. of cerebrospinal syphilis occurs within the first two years.

In addition to this variety, there are a few cases, recently described, which seem to show that there is a true syphilitic encephalitis analogous to syphilitic myelitis. These cases will be referred to later.

Symptoms.

The symptoms of syphilitic encephalitis do not constitute an individual complex. It is for this reason that we refer to the subject in

the briefest possible manner. In the diffuse form the symptoms will depend very largely on the seat or location of the pathological process, and to a much less extent on the size. If the lesion is of the brain substance around the central fissure, motor irritation symptoms, as twitchings, epileptiform attacks, or monoplegia (partial or transitory), aphasia, etc., will be the predominant symptoms. In short, the symptoms of this form of brain syphilis do not differ materially from those of gumma or other tumors of the brain, except by the relative severity of the irritation symptoms, the absence of pressure symptoms, and the frequency and severity of mental symptoms. It is the lesion, in all probability, that is most often the organic basis for syphilitic epilepsy, although meningoencephalitis syphilitica, when of the motor convexity, and cicatricial tissue at the site of resorbed previous gummatous formation and deposit, are frequently the anatomical lesions of syphilitic epilepsy. I speak now of the epilepsy of acquired and not of inherited syphilis. The epilepsy of congenital syphilis may be the expression of some dyscrasic condition or of an inherited gummatous process in the cortex or the meninges.

Some of the peculiarities of syphilitic epilepsy must be referred to here, although they will be considered in greater detail in the chapter devoted to epilepsy. In a general way, it may be said that epilepsy which develops after well-advanced maturity, after the end of the third decade of life, without history of trauma or neuropathic heritage, in a temperate individual, even though a statement of an initial lesion cannot be elicited, and the occurrence of secondary manifestations is denied, is almost always of syphilitic origin. If the primary lesion or secondary manifestations of syphilis can be established in such a case, the luetic origin of the epilepsy is beyond cavil, and the disease should be treated from that standpoint.

It is usually taught that syphilitic epilepsy is partial or Jacksonian, and such teachings are in large measure true. The labors of Fournier, of Kowalewsky, and of others, have shown that oftentimes the clinical manifestations of syphilitic epilepsy are not detectably different from those of true epilepsy, that is, so-called idiopathic epilepsy existing from early youth. The last-named writer believes that the attacks of epilepsy will be cortical or Jacksonian when the syphilitic process causing it is an isolated gumma, irregular gummatous process, or scar left after the absorption of previous exudation. When the epilepsy manifests itself as idiopathic, or, as he calls it, "medullary" epilepsy, it is due to the influence of infection of the organism by the contagion of syphilis. Be this as it may, there are certain symptoms associated with syphilitic epilepsy which are suggestive enough to warrant putting the patient on vigorous an-

tisyphilitic treatment. The most important of these, aside from the general facts mentioned above, is the occurrence of partial epilepsy with considerable preservation of consciousness. That is, a leg, or an arm, or some other part may become convulsed, with or without preceding sensory aura, and yet the patient suffers only a slight obscuration of consciousness. If a history of such attacks be obtained, and the patient at the time of the examination has attacks with complete loss of consciousness, suspicion of the syphilitic origin of the disease should be aroused. Another important symptom is the condition of the patient, both in mind and body, between the convulsive attacks. In syphilitic epilepsy the patient is not free from symptoms, such as headache, depression, lethargy, bodily sluggishness; while the patient with ordinary epilepsy, if not depressed from bromide administration, is quite well between attacks. The paralysis, paresis, monospasms, or contractures, that often follow convulsive attacks in syphilitic epilepsy, have no particular diagnostic value, except that their *bizarrerie* and association, and the fact that no other cause can be predicated save an anterior infection, suggest syphilis. These symptoms, however, merely signify some cortical destruction, which may be due to any neoplasm other than syphilitic. The same may be said of nocturnal attacks—they have no significance for or against a syphilitic basis of the epilepsy. If all possible causes of epilepsv can be eliminated in an adult, and if the administration of antisyphilitic remedies seems to benefit the patient, it is certain that the epilepsy is of a luetic nature.

The mental symptoms of a patient with syphilitic encephalitis are a very variable quantity. In one instance the striking symptom may be such a progressive weakness of all of the mental faculties as to constitute dementia; while in another the prominent shortcoming may be defective inhibition, which reveals itself by moral misconduct, misdemeanors, and crimes. Occasionally, meningoencephalitis syphilitica may almost exactly simulate the symptoms of general paresis. Not infrequently acute maniacal outbursts or continued mania may be the form which the mental disorder takes. The rapidity with which the mental symptoms change is noted as characteristic. In many cases there are no suggestive subjective complaints and no characteristic objective phenomena. The diagnosis must be made by exclusion.

Disseminated, circumscribed syphilitic encephalitis, such as is described by Charcot and Gombault, does not cause a symptom complex which can be diagnosticated as syphilitic. Its occurrence is rare, and then almost always in children with hereditary syphilis. In some cases, no doubt, it produces the cerebral type of disseminated

insular sclerosis, but even then the syphilitic nature of the disease can only be suspected by reason of a history of previous infection and secondary manifestations, or of the presence of other more unerring syphilitic accompaniments.

A form of acute brain softening of inflammatory origin, which occurs independently of disease of the blood-vessels, and which is due to syphilis, has been described by Jürgens, Oppenheim, Keller, *et al.* In the cases they describe the symptoms were those of thrombotic hemiplegia; in the case of Keller the symptoms were typically those of thrombosis of the basilar artery, yet after death the pathological changes were confined to foci of inflammatory softening in two separate areas of the pons, the blood-vessels being free from degenerative disease. In Jürgens' case the apoplectic attack, hemiplegia, and coma came on while the patient was in the secondary stage of syphilis. These cases have nothing pathognomonic about them, and, like those in which patches of sclerosis develop, they can only be suspected.

The subject of syphilis of the nervous system leads me to say a word about the classification of nervous diseases, for, although it may seem at first sight not in keeping, it is entirely apropos. The present nosology of the diseases of the nervous system is a most unsatisfactory one, and particularly because there is no spirit of agreement among writers. The classification that has the greatest number of followers is the pathological one. We are gradually receding from it, however, and drawing nearer to the only satisfactory systematic classification (so it seems to the writer), one based on etiology. When nervous diseases are classified nosologically, according to their causation, we shall speak of infectious nervous diseases, diathetic nervous diseases, syphilitic nervous diseases, toxic, autotoxic, traumatic nervous diseases, diseases of defective development, etc. Such a classification has already been espoused by many latter-day writers, and we believe to an elucidatory advantage. It is particularly the nervous diseases due to syphilis, however, that have been singled out for this classification, so that to-day the expressions syphilis of the nervous system and cerebrospinal syphilis are used in every-day scientific parlance, and this is my reason for emphasizing the fact that the use of such terms does not carry with it any pathological entity or type. Syphilis causes the vast majority of organic nervous diseases occurring in adult life, which are not the result of two factors—infection and defective development. If the truth of this be granted, there is no need of apology for giving extensive space to the subject. In this treatise syphilitic diseases of the brain are not considered as clinical entities, but are considered under the

diseases which syphilis causes: Syphilitic meningitis in the section on Meningitis; syphilitic disease of the blood-vessels under Apoplexy; parasyphilitic corticomeningeal diseases under General Paralysis, circumscribed gummata under Tumors, etc.

This shall not prevent us from referring briefly to the various forms of syphilis of the nervous system, and of saying a few words concerning general etiology and pathogenesis.

Syphilis of the nervous system must be divided into syphilitic and parasyphilitic. Under the former may be considered: Syphilis of the intracranial contents, syphilis of the intraspinal contents, cerebrospinal syphilis, and syphilitic diseases of the nerve trunks. Under the latter, tabes dorsalis, general paralysis, syphilitic neurasthenia, syphilitic neuralgia, and a form of epilepsy. True hysteria, I believe, is never of syphilitic origin.

Syphilis of the intracranial contents may be subdivided into syphilis of the meninges, of the brain substance, and of the blood-vessels, it being understood that any part of the meninges or any constituent of the encephalon may be the seat of the lesion, and that all these conditions may occur simultaneously. Syphilis of the intraspinal contents may be subdivided in exactly a similar way, with the addition that a form of syphilitic transverse myelitis has been raised by Erb and many other writers to the dignity of a separate disease, to which the name syphilitic spinal paralysis has been given.

The term cerebrospinal syphilis is given to a diffuse gummatous meningoencephalitis and meningomyelitis, the basilar portion of the brain and the upper part of the spinal cord being specially predilected as the seat of the exudation and subsequent degeneration. It is usually evidenced clinically by symptoms of cranial nerve affections, of hemiplegia, of a spastic type of spinal paralysis, and by one or several distinct remissions. A history of infection can usually be obtained.

The period of time that elapses between syphilitic infection and exudative manifestations of syphilis in the nervous system was formerly thought to be much longer than we now know it to be. I have already referred to this point, and may again say that the liability to luetic nervous disease is immeasurably greater during the first ten years succeeding infection than in all the other years of the infected person's life, and that the liability during the first five years after infection is much greater than during the next five.

The proportion of cases of syphilis that are followed by symptoms indicative of lesion within the skull, conditioned by the anterior syphilis, is very much greater than syphilographers as a class are willing to admit, and greater than is usually taught in the text-books. Hjelmann believes that about twelve per cent. of all patients with ter-

tiary syphilitic disease show symptoms of brain syphilis, and Fournier puts the percentage much higher—twenty-one per cent. My own belief is that the estimate of the former writer is scarcely high enough. The factors that predispose to luetic disease of the brain substance, following on previous infection, are the same as those that have been enumerated as contributing to other forms of syphilis of the nervous system—for instance, syphilitic meningitis, the most important of which are excesses of all kinds.

Affections of the nervous system which are attributed to syphilis are of at least two very distinct pathological natures—the exudative and degenerative. It does not follow that the two are not often combined, and, as a matter of fact, the exudative variety is generally followed by profound degeneration. The degenerative variety, however—that is degenerative *ab initio*—is preceded by the ordinary changes of exudation and proliferation; nor are the lesions found after death macroscopically or microscopically at all characteristic of a luetic process. The classification of the diseased states arising after and from syphilis, as specific and parasyphilitic, which includes under the first those which are the direct result of the syphilitic action, specific processes, and lesions of syphilis, and under the latter those which are the products of the action of the syphilis, but are not of specific nature is a serviceable one. In short, the parasyphilitic diseases are syphilitic diseases whose anatomical basis is a degenerative one from the beginning—such as tabes, general paresis, and some forms of epilepsy, and Fournier includes also a form of neurasthenia and hysterosyphilis.

The hereditary parasyphilitic diseases are exclusively defects of development, and what little is known of them is referred to in the section on that subject.

When the term syphilis of the nervous system is used, therefore, it may cover a multitude of very differently located pathological processes, and no excuse for the use of such a nomenclature to indicate a particular lesion or an individual pathological change can be put forward. The only generic term of this kind that is warranted is multiple cerebrospinal syphilis, which stands for a diffuse, widespread, gummatous meningitis of the brain and spinal cord. The substance of the brain and cord may be invaded, but in all probability the latter is secondary to the former, and never occurs without it. Multiple cerebrospinal syphilis constitutes a pathological and clinical entity, and the symptomatology is that of syphilitic meningitis, plus that of syphilitic encephalitis and myelitis, in varying and constantly changing quantity. This subject is treated of in the article on Diseases of the Meninges in the present volume.

Diffused Sclerosis of the Brain.

The occurrence of diffuse sclerosis of the brain as well as of the entire central nervous system—using that term in the sense of its derivation, and not in a pathological sense—implying an increase in the sustentacular tissue of the brain, cannot be denied. What the exact pathological process is that goes on in these cases we are unable to say, because the post-mortem findings are not the same in all cases. In some cases, the majority perhaps, the principal change is in the interstitial tissue, of the nature of neuroglia and connective-tissue proliferation and overgrowth, while the ganglion cells and the nerve fibres are but slightly affected. In other cases there are in addition to alterations in the blood-vessels unmistakable evidences of previous inflammation and such indicators of atrophy of the nerve substance as the presence of amylaceous bodies in considerable numbers. In all cases there is an increase in the consistence of the brain or in those parts of it in which the sclerosis has gone on, and in some cases there is diminution in size of the brain. Those instances in which the volume of the brain is materially diminished are considered by some writers as forms of atrophy of the brain, but universal atrophy of the brain occurring as a primary condition is a pathological rarity; and as for those varieties of brain atrophy that are secondary to some primary diseases, they should not be considered individually, but merely as an anatomical sign post of the primary disease. The microscopic appearance of sclerosis of the brain occurring after chronic inflammation and that occurring from degeneration without inflammation is the same in both instances. If attempt at distinction be made between these two conditions, it is that the first is not so universally diffuse as the second, although it is essentially a diffuse lesion. Diffuse sclerosis of the brain as a disease entity is of extremely rare occurrence. I have had opportunity of observing but one case. The subject has been contributed to particularly by Erler, Berg, Schmaus, Buss, and Burk, and it is to the articles by these writers that I am indebted for the brief description herewith given.

It occurs as a terminal stage of other conditions—for instance, inherited syphilis in infants, around congenital and acquired defects such as porencephaly and spots of softening. It is not at all of these latter forms that we speak, but of widespread sclerosis developing apparently as a primary condition, which is attended by an increase of the connective-tissue support, not necessarily secondary to a diminution of the nerve elements, nor attended by it in the beginning to any marked degree.

To the naked eye the brain as it lies *in situ*, after removal of the calvarium, seems in many cases to be separated from the dura by a space varying in depth up to a half-inch. The brain preserves its normal contour, but may give the appearance of being slightly flattened on the convex surface of the hemispheres; rarely is it diminished in size. Its increased consistence is quickly apparent on removing it from the skull cavity. In some cases, on stripping off the pia, this will be found thickened or adherent. When it is detached the sulci and fissures may have a normal appearance, but frequently they are more widely separated than normal and somewhat gaping. After the pia is removed the hardness of the brain is still more striking. It does not alter its shape or fluctuate as its position is changed, nor on handling. On cross-section the leather-like consistence resists the knife, which passes through it with a creaking sound. When the ventricles are cut through, particularly the lateral, the cavities may appear to be enlarged, but actually they are rarely so, the appearance being due to the fixity of the roofs and walls.

The white substance is the principal seat of the sclerosis, which may be more extensive and widespread in one hemisphere than in the other; the cortical substance is generally thinner than normal, and this often gives the convolutions an atrophic appearance. In the cases referred to above as occurring in young children affected with inherited syphilis, the gray matter is more distinctly the seat of the sclerosis and it is sometimes limited to one or more convolutions, while the white substance is affected to a much less degree. In these cases too the atrophy of the brain is always very considerable. In a case reported by Buss the morbid conditions were porencephaly, atrophy, and induration of the convolutions. In Schmaus' case also there was extreme cerebral atrophy, internal and external hydrocephalus and sclerosis of many gyri, the changes being confined principally to the neuroglia.

There is every reason to believe that diffuse sclerosis may follow after acute non-purulent encephalitis. In such cases the sclerosis will be associated with vascular changes, most pronounced in the cortex, and the microscope will reveal the proliferation in the connective tissue, which goes on hand-in-hand with the disappearance of nerve substance. In this category I place such cases as the one reported by Knaggs and Brown, in which the symptoms were those of infantile cerebral palsy, due to acute non-purulent encephalitis. After death the right hemisphere was atrophied, the layers of the cortex were unrecognizable, and the cortex itself was devoid of all trace of nerve structure, showing principally condensed neuroglia, and post-inflammatory sclerosis.

The primary pathological condition in this disease and in many other degenerative diseases will not be revealed until a more perfect technic for the staining of the neuroglia shall have been found. (How far Weigert's most recent method accomplishes this cannot yet be said.) No further statements can be made as to the etiology of diffuse sclerosis than are implied in the remarks anent its pathology. In one case (Strümpell) chronic alcoholism was the supposed cause. In some a remote syphilitic infection has been mentioned (Johnston), while in other cases (Wespthal, Jolly, and others) it has developed in connection with disseminated insular sclerosis, the mass of diffuse sclerosis blending with several foci. Leaving out the cases occurring in syphilitic infants, the disease is one of adult and advanced life, it often being associated with sclerotic changes in the hæmatopoietic organs. Its duration is as indefinite as its etiology, a case having recently been reported (Burk) in which it lasted through half a century.

A sufficient number of cases are not on record to warrant positive statements as to the symptomatology. From the nature and location of the lesion, however, it is plain that the symptoms are paralytic and irritation manifestations in the motor sphere. There may be twitchings or tremblings in the extremities, of the character of intention tremor, or there may be attacks, apoplectiform and epileptiform, or spontaneous and compulsory movements. The paralytic manifestations are paresis of the extremities, of the seventh nerve and the eye musculature (rare), and in the advanced stages of the disease, incontinence of urine and fæces. In addition to these there are not infrequently some hindrance of speech and difficulty of swallowing. Psychical functions are occasionally impaired early, but in some cases in which widespread sclerosis was found after death, disorder of intellect was absent or of such a slight degree as to in no way interfere with the patient's capacity to care for himself. In some cases there are slight sensory disturbances, in the shape of a mild degree of anæsthesia, but paræsthesiæ have not been reported. In many cases there is an increase of myotatic irritability and an exaggeration of tendon jerks, but in others the reflexes are lost.

In syphilitic children the diagnosis will be suggested if in addition to somatic evidences of that disease there are mental shortcomings, varying from slight psychical disturbance up to complete absence of mental activity, depending upon the extent and progress of the cerebral lesion, in association with some or all of the symptoms already given, such as convulsions, contracture of the extremities, increased myotatic irritability, nystagmus, etc. The more uncommon symptoms of diffuse sclerosis, pupillary changes, nystagmus,

intention tremor, paralysis of the eye muscles, and headache, may all be present.

Nothing pertinent can be said about the prognosis or treatment of this disease, as it is extremely improbable that the diagnosis can be made with any degree of certainty during life. If it were made the therapy indicated would be the administration of such remedies as have a deserved reputation of being at least inimical to proliferation of neuroglia and connective-tissue overgrowth, such as silver and arsenic, the removal of all conditions that contribute to bodily and mental irritation, and the increase of the patient's nutrition.

It needs no special mention to emphasize what is universally conceded, viz.: that in children who have signs of inherited syphilis the administration of small doses of mercury is the only measure of any therapeutic utility.

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INFANTILE CEREBRAL PALSIES.

Infantile cerebral palsy is the clinical designation of a distinct class of cases occurring in young children. Anatomically and pathologically the clinical complex is dependent upon widely different conditions, but all of them have in common a degree of paralysis varying from the mildest paresis to the completest abolition of voluntary movements, from which the condition takes its name. In fact, it may be said that all inflammatory (with the exclusion of the forms excited by pyogenic organisms), degenerative, and hemorrhagic conditions in the brain, be they prenatal, natal, or postnatal up to the time of second dentition, furnish the anatomical basis of the infantile cerebral palsies. In this section, therefore, many different pathological conditions which are attended by a more or less clearly defined clinical entity must perforce be considered. It includes teratological and defective protal states, states of arrested development, conditions due to injury and disease mediated through the mother to the child in utero, as well as the many traumatic, inflammatory, and degenerative defects of the brain which may occur in the first decade of a child's life.

It would be quite possible to elevate a number of the morbid conditions included in the description to the dignity of individual considerations, and speak separately of their etiology, pathology, and evolution; in fact, some of them have been considered under meningitis and meningeal hemorrhage, encephalitis, hemorrhagic and nonpurulent, and as the anatomical basis of a species of epilepsy and idiocy. But the clinical picture of infantile cerebral palsy, it matters not what the pathogenesis of it is, does not vary in the essential features; the symptoms differ in amount, in intensity, and in combination. It therefore contributes to a readier understanding of the symptoms to consider infantile cerebral palsies as a clinical entity.

History.

Our knowledge of the infantile cerebral palsies is to a greater extent the result of labors of the present generation of neurologists and pathologists than is that of any other organic disease of the central nervous system, with the exception of syringomyelia. Naturally, writers whose works are now considered ancient were not ignorant of their existence nor unmindful of their occurrence, and some of them have left records of fairly true clinical descriptions, but as Freud and Rie have pointed out, Cazauveila was one of the first, if not the first, to describe types of these palsies in relation to the pathological conditions found after death. The date of his work is 1827. The next most important work on the subject was a graduation thesis, that of Henoch, "De Atrophia Cerebri," in which brain conditions associated with infantile cerebral hemiplegia were carefully described. Cruveilhier did not fail to notice and depict some of the morbid states in that atlas which still excites the admiration of pathologists. The most important work on the subject, however, after that of the first named, was that of a compatriot, Cotard, who, in 1868, paid special attention to the pathological anatomy of this symptom complex. Many of the findings and of the interpretations put upon them by this author as well as by another pupil of Charcot, Turner, are still accepted by neuropathologists. Although the work of Cotard became widely known, very little was contributed to the knowledge of these conditions for the next decade, with the striking exception of the clinical observations of Little. For many years previous to this time the English surgeon had studied and described, but apparently without recognizing their true pathological nature, physical and mental shortcomings occurring in children born in premature, difficult, and complicated labor, especially in relation to the deformities of a spastic nature that occur. With the individual com-

plex of symptoms which this author described his name has long eponymically been associated. At the present time Little's disease is recognized as a variety of the class of disease which we are about to consider. It is often referred to as congenital spastic rigidity or spasmodic tabes. In the mean time, many German authors such as Seeligmüller, Wuillamier, had given the pathology of the affection careful study. In 1882, two very important contributions were made, one by Visioli, the other by Kundrat, which materially broadened our grasp of this disease. As has been shown by Freud, the former writer antedated Strümpell, who some years later not only popularized one view of the pathology of infantile cerebral palsies, but contributed remarkably to our conception of encephalopathies, by advancing the theory that the lesion was in many cases an acute inflammation, in short, was an acute polioencephalitis. Kundrat differentiated most lucidly congenital and acquired cerebral palsies from an anatomical standpoint and suggested the term porencephalia, or at least popularized it; for Heschl had previously used it to designate a condition of the hemispheres characterized by hollows, mostly on the surface, and due principally to loss of substance the result of hemorrhage, thrombosis, or embolism, occurring during intrauterine life or at the time of birth. A year later Audry made an important clinical contribution, basing his results on an analysis of one hundred and three cases. About at the same time Ross had given careful consideration to the disease, not alone in his treatise on nervous diseases, which appeared in 1881, but also in an article in *Brain*, a year later. In America, the writings of Sinkler, of Hammond, and of McNutt did much to disseminate a knowledge of the affection.

It is impossible to enumerate individually the writers at home and abroad who have materially assisted in establishing the various forms of the cerebral palsies on a firm pathological basis during the past decade. We must content ourselves by saying that on this side of the water they include the names of Sachs, Peterson, Fisher, Lovett, and Osler; in Germany, Freud and Rie, Kundrat, Strümpell, and Henoch; in France, Marie, Bourneville, and Déjerine; and in England, Gowers, Boyd, and Thompson. The credit of having demonstrated a familiary type of the spastic palsies is due to Sachs, who in 1887 described this form of the disease, which has since been universally recognized.

Etiology.

Before enumerating the specific etiological factors it may be well to attempt a classification of the different forms of infantile cerebral palsies. It is readily seen that such a classification can be built

up according to the etiological factors, according to the pathological conditions, or according to the clinical manifestations. In fact, we shall have to consider each of these three forms, and in this place we shall refer to the etiological factors according to the time when they exercise their injurious effects, that is, whether on the proton of the fœtus, the fœtus, the child at birth and after birth, or through the mother. There can be no doubt that the somatic basis of this disease originates contemporaneously with the development of the primary constituents of the fœtus, and that such developmental defects are the indirect results of pathological heritage. The morbid conditions that are operative on the child during its prenatal life may be mediated through the vascularity of the brain, and consist of intrauterine inflammation of the brain, softening, disintegration, and atrophy, all of which may follow rupture or occlusion of a blood-vessel. The fact that some cases of infantile cerebral palsy occur in this way is certified to by the report of a case observed by Osler. In the uterus of a young woman who had died of typhoid fever there was found a six months' fœtus, the left hemisphere of whose brain showed a striking condition of porencephalia. The newly formed cavity had ragged, irregular walls, and contained a large recent clot, which had ruptured through the basal ganglia into the lateral ventricle. It is probable that not only do the infectious fevers which may attack the fœtus during its intrauterine life, cause many congenital birth palsies in this way, but syphilis as well, the first by leading to embolism and thrombus of the blood-vessels of the brain, and the latter to thrombus and rupture. Some recent authors, such as Sachs, are not willing to concede that hereditary syphilis plays any prominent part in the etiology of intrauterine palsies. But if the somatic accompaniments which ordinarily attend inherited syphilis, such as Hutchinson's teeth, ulceration of the cornea, parenchymatous keratitis, acrid nasal discharge, defective osseous formations such as of the bridge of the nose, are to be taken to indicate inherited syphilis, the writer's individual experience based upon a study of forty-three cases seen in the clinic of Professor Dana, and in his own service in the Charity Hospital, leads him to the opinion that inherited syphilis must be considered one of the etiological factors, for its influence was evident in no less than five cases.

The most important etiological factors acting through the mother are those of direct and indirect trauma, including under the latter the injuries that may result to the fœtus from injury, of any origin, to the mother. Accidental and purposely inflicted blows on the mother's abdomen during the latter months of the child's intra-uterine life, even though they do not cause premature birth, are sometimes

sufficient to give rise to cerebral palsies. It is possible that acute infectious diseases of the mother may produce circulatory disturbance in the brain of the child, and in this way be the exciting cause of some brain lesion. We do not believe that fright, anxiety, mental worry, grief, and the like, exercise any contributory influence, except that they may impair the mother's nutrition, which impairment may react unfavorably upon her uterine content.

The causes that are operative at the time of birth and after birth are immeasurably more frequent and more important than those just mentioned. Statistically, it has been proven that the large proportion of cases occur between the period of birth and the end of the third year. It is unnecessary to enumerate all the causes that may be contributory to the encephalopathies at the period of natality. They may be summed up in one word, and that word is dystocia. It is probable that the liability to occurrence of injury of the brain at the time of birth is in direct proportion to the duration of labor. Severity of labor and artificial means to facilitate it are not such important factors as were at one time supposed. It would seem that the conditions which cause asphyxia are also favorable to the production of birth palsies. The reason of this is not difficult of interpretation. Asphyxia causes a retardation of the circulation, the injurious action of which is soon manifested in that organ, the brain, which is not well supplied with collateral circulation; and the consequences sometimes are that the vessels become plugged or ruptured. In some cases the circulatory disturbance is manifested also in the cord, either associated with lesion of the brain or apart from it. In this way are explained the few cases of birth palsies which are due to primary lesions of the cord. Examples of this have been cited by Spencer.

Postnatal birth palsies may be due to causes operative (1) on the blood-vessels to produce hemorrhage, embolism, or thrombosis; (2) on the blood-vessels and structural tissues of the brain to produce encephalitis; and (3) on the meninges of the brain to produce inflammation and hemorrhage. The acute infectious diseases, influenza, diphtheria, typhoid fever, scarlet fever, and the like, may produce infantile palsy in either of the two ways first mentioned. Blows on the head, pyogenic diseases of adjacent skull cavities, and acute infectious diseases may be the antecedent factors producing meningeal hemorrhage, sinus thrombosis, and meningitis.

It has been said above that Strümpell disseminated and popularized the idea, particularly among the French school of neuropathologists, that the majority of the cases of postnatal cerebral palsies were dependent upon inflammation in the brain, particularly

of the gray matter, analogous to the lesion in anterior poliomyelitis and for which he proposed the name of polioencephalitis. These views of Strümpell and Marie have been accepted to a certain degree by many neurologists; but this does not mean that all cases of infantile cerebral palsies, nor even a majority of those occurring after birth, are dependent upon inflammation. We are led to this opinion from clinical experience alone; death occurs so rarely during the active period of the disease that when the brain comes under observation, perhaps many years later, it is impossible to tell whether there has been acute inflammation antedating the degenerative changes or not. It must be admitted, however, that the sudden development of a unilateral paralysis, preceded or attended by a series of convulsions, considerable elevation of temperature, and a more or less prolonged period of unconsciousness, as many of these cases are, is fairly tangible evidence for considering some of the infantile cerebral palsies to be of inflammatory origin, particularly in the light of our present knowledge of the causation and pathology of acute anterior poliomyelitis, and acute non-suppurative encephalitis. Conceding this, however, it does not by any means follow that a proportion of cases of birth palsy are not due strictly to hæmic and vascular causes. There should be no controversy on this point. Morbid conditions of the blood predisposing to coagulation may occur in infancy, as may also defective structural changes in the blood-vessels which lead to rupture; and it is just as unscientific to contend that genuine cerebral apoplexy does not occur in infancy because the latter is a disease of old age as to claim for the same reason that cirrhosis of the liver is not sometimes seen in childhood. Sachs and Déjerine have both shown conclusively that the lesion in the brain in cases that have come to autopsy within a reasonably short time after their occurrence, is that of hemorrhage, areas of ischæmic softening, or a hemorrhagic equivalent such as a cyst. I believe, however, that the larger number of cases of postnatal cerebral palsies of infancy are the result of inflammation rather than of degeneration pure and simple, that is, without antecedent inflammation. At the present stage of our knowledge of the familiary form of infantile palsy it is impossible to say anything definite of the pathological genesis of the disease. Sachs, who was the first to describe this condition, and who has since contributed to its elucidation, inclines to the belief that these cases are certainly not of inflammatory origin. This belief seemed to receive support by one of his cases that came to autopsy, although sufficiently convincing evidence was brought forth to show that an anterior inflammation had not existed. There are, however, certain peculiarities in the clinical history of the familiary forms of

infantile palsy which incline one to believe that the lesion is degenerative *ab initio.*

Symptoms.

The symptoms of infantile cerebral palsy may be divided into the motor symptoms, the sensory symptoms, and the psychical symptoms, the first and the last being by far the most important. The motor symptoms are some degree of paralysis, contracture, convulsions, athetosis, compulsory, associate, or choreiform movements. The psychical symptoms are certain limitations in mental development, and forms of intellectual expression varying from the slightest sluggishness in reaction to the environment up to complete idiocy. The cases that present sensory defect are comparatively few, and it is not at all certain that such symptoms, even when they can be made out, should be divorced from intellectual shortcomings. They consist of disturbance of the various special senses, particularly of sight, of hearing, and of the tactual sense. The paralysis may assume the form of diplegia, of hemiplegia, of paraplegia, and possibly of monoplegia. The latter is probably very rare, although it is not improbable that some cases of central facial palsy, of brachial palsy, and of congenital clubfoot are manifestations of this disease. Diplegia is the most common form in which prenatal cerebral palsy manifests itself, and hemiplegia the most frequent form for the natal and postnatal varieties. In 225 cases analyzed by Sachs and Peterson, hemiplegia was the form which the paralysis took in 156 cases, and it was nearly equally divided between the left and the right side, the preponderance being a little in favor of the right side, as it is in adults. In my own cases, hemiplegia occurred in 29 cases, diplegia in 9, paraplegia in 3, and monoplegia in 1. The paralysis is almost always followed and attended by contracture, and for this reason is frequently described as spasmodic hemiplegia, diplegia, or paraplegia. Symptoms of motorial irritation manifesting themselves by athetosis and simple or spasmodic choreic movements, and epileptiform convulsions are rarely absent.

Those cases of birth palsy that are classified on a temporal basis as prenatal and natal have a different symptomatology than the postnatal—in fact, it might be said that the occurrence of the former is without symptoms, for they take place at a time when the physician cannot be cognizant of them. Their later accompaniments, however, are not materially different from those of the postnatal form.

The symptoms of the latter forms develop in a fairly constant manner. At any time between birth and the period of second dentition, but more particularly before the third year, the infant may be-

come restless and feverish, and then suddenly be seized by a convulsion or a series of convulsions manifesting themselves on one side of the body or over the whole body, which are soon followed by a period of more or less profound unconsciousness. Febrile manifestations continue during the unconsciousness, and the abatement of the fever is generally coincident with a gradual return to consciousness. At this time it is discovered that the patient is partially or completely hemiplegic, diplegic, or paraplegic. Occasionally the paralysis develops gradually after consciousness is partially restored, or it may be noted to progress during the unconsciousness. Sometimes the paralysis comes on in attacks, it being noticed that after each convulsive attack the paralysis is more complete or more extensive. The most common distribution of paralysis is to one side of the body, the face, especially the upper part, being also affected. Generally, the intensity of the hemiplegia is greatest in the upper extremity, and as in cases of apoplexy of the adult signs of improvement appear last in this part. The paralysis at first is flaccid and often complete, totally devoid of voluntary movement. In very young children it may pass for a time unnoticed, and for this reason in dispensary and hospital practice it is difficult often to elicit the exact date of onset of the paralysis and its temporal relation to convulsions. In my individual experience, initial convulsions preceding the paralysis have been the rule, and it is not usual for paralysis to become complete without their occurrence.

After a time, varying from two to four weeks, rarely longer, evidences of descending degeneration in the motor tract and destruction of the inhibitory influences of the brain on reflexes begin to show themselves in the shape of contractures, ankle clonus, exaggerated knee jerks, and increased myotatic irritability of the paralyzed side or sides. These contractures are principally of flexor muscles and are particularly manifest in the terminal parts of the extremities, and often thus cause striking deformities, club-foot, claw-hand, etc. (see Fig. 13). The hands and forearms become flexed and pronated, or sometimes the fingers and hands go into a position of forced extension. The extent and intensity of the contractures may become so great as to cause extremest deformity. In the beginning the spasmodic state of the muscles of the paralyzed parts is mobile, but this disappears usually in proportion to the duration of the condition and the intensity and rapidity of secondary degeneration. Contractures develop in many cases, especially in children, with surprising severity and completeness. During their development they are often accompanied by great pain, the result not only of cramp in the muscles, but of enforced positions. As the child ages the paralyzed side grows, but not *pari*

passu with the unaffected side of the body (Fig. 14). There is no degenerative atrophy or suggestive elicitation of it, such as perversion in the response to electrical currents. If the paralysis is of the diplegic variety, some retardation of development on both sides is evi-

FIG. 13.—To Illustrate Particularly Deformity of the Feet.

dent. The only other trophic disturbances that have been noticed are in the genital organs of the male and the mammary glands of the female at the time of puberty. Such conditions have been noted by Leblais, who also remarked that occasionally pubic hirsuteness was less abundant, and the testicle was smaller on the paralyzed side. It has also been noted that the nails grow faster on the hand and foot of

the paralyzed side. The vasomotor tone of the paralyzed side is low, and this is responsible for the lifeless feel that the parts often have. Almost never are sensory shortcomings present.

The next most common symptom or manifestation of involuntary movements depends upon the kind and seat of the lesion. If the

FIG. 14.—To Illustrate Difference in Size of the Two Lateral Halves of the Body.

trouble be essentially of the blood-vessels, the lower levels of the central nervous system, the internal capsule, and the midbrain will likely be the seat of the lesion, and athetosis manifested in one or both sides of the body will be, next to the paralysis, the most striking symptom. Naturally this does not imply that athetosis may not result from cortical lesion. In fact, in the bilateral form of athetosis, a much

more uncommon symptom than unilateral, it is probable that at the onset the processes that call forth athetoid movements are cortical and subcortical.

The athetoid movements of the post-natal infantile cerebral palsies occur in the fingers and hand of the affected side, and clinically differ in no way from the athetosis of adult hemiplegia. A condition of advanced contracture, or in fact any considerable degree of contracture, is inimical to the rhythmical coördinate movements of athetosis. It need not, therefore, be insisted upon that cases in which hemiathetosis is a prominent symptom are not attended by the development of secondary degeneration, permanent spastic condition, and its entailments in a severe form, such as increased myotatic irritability. In cases in which athetosis is present, the paralysis is of a degree which often allows of the coarser voluntary movements, but all movements of purposive finesse are lost. The affected side of the body is mobile, clumsy, and carried in a rigid manner, which causes considerable deformity. Associated movements occur next in frequency to athetoid movements. They are most frequently manifest in the hand and vary in intensity and volume from the slightest associate movement when the normal side moves, up to complete imitation. Associate movements are most common in infantile cerebral palsies of the hemiplegic type. Other involuntary movements which the paralyzed limbs sometimes show are those designated as choreic. Choreic movements occurring with cerebral palsies of infancy are in all probability oftenest associated with cortical lesions, although they may be called forth by lesions situated at the level of the internal capsule. They occur with similar somatic changes of paralysis, contracture, etc., as athetosis, and can readily be distinguished from the former by their *brusquerie*, their lack of rhythm and purposiveness, and by their temporary cessation.

Choreic movements are more likely to be seen in those cases of infantile cerebral palsy that appear after the third year than in those occurring soon after birth. Reckoned from the time of the onset of the disease, they are a comparatively late manifestation; a number of cases being on record in which they occurred some years afterwards. This fact alone, and particularly if they are first remarked after a fright, sometimes causes them to be confounded with chorea. The differential points in the diagnosis of these three conditions will be considered later.

The symptoms or phenomena which are next to develop are the motor or sensorimotor convulsive phenomena. These may assume the form of Jacksonian epilepsy, of half-sided epilepsy, of severe double-sided epilepsy, or of petit mal. The attacks of fits tend to

increase in frequency, in severity, and in extent of motor implication as time goes on. They predicate the existence of cortical and subcortical lesions, which are followed by consecutive descending degenerations in the motor tracts, and upon this degeneration the associated contractures are dependent. The time of appearance of epilepsy is a variable one. In some cases a condition of status epilepticus precedes the paralysis. Indeed, some authors have gone so far as to say that the epileptic convulsions are often the determining factors in the causation of the paralysis. For instance, Sachs says in many cases the palsy seems to be a more or less immediate result of the convulsive seizure. It is probable that the convulsions which precede the paralysis in a number of cases are not strictly of an epileptic nature, that is, the pathological basis of the convulsions that precede and those that follow the paralysis is not the same; that the two are expressions of radically different forms of injury. Be that as it may, however, it is impossible to exclude the pre-paralytic convulsive attacks from the clinical category of epilepsy. König relates a case developing at the seventh year in a child who had had epileptic convulsions from the time it was ten months old. It is not unlikely that a part, at least, of the cases in which convulsive attacks precede the paralytic phenomena are associated with meningeal hemorrhage and meningoencephalitis. In the cases that I have had under personal observation it has frequently been noted by the parents that in the beginning the paralyzed side was most severely convulsed. This has often suggested to ignorant mothers the possibility that the epilepsy was not an undesirable occurrence as it made the paralyzed parts move. After the first attack of epileptic convulsions the second, and even the few succeeding it, may be delayed for a considerable time, but there is nothing quite so certain in clinical neurology as the fact that they will increase in frequency and severity as time goes on. Twenty and thirty severe attacks a day may be the unfortunate quota. I have seen a patient have no less than four severe attacks while waiting her turn in the outpatient department. After the epilepsy has persisted for some time, there is nothing in its outward manifestations to distinguish it from what we empirically call idiopathic epilepsy. In fact, it may be possible that some cases of the latter disease have their genesis in an early and slight attack of infantile paralysis in which there has been such a degree of spontaneous recovery as to make its previous existence doubtful. Very few cases, however, will fail to reveal their true origin when subjected to a critical routine examination. There will be some slight defect in the finer coördinate movements subserving dexterity and digital purposiveness, a slight amount

of rigidity, of ankle clonus, or an excess of knee jerks, or even a trace of deficient innervation in the upper part of the face supplied by the superior branches of the seventh nerve. It must be remembered that epilepsy is not necessarily a part of the clinical picture of infantile cerebral palsies. Freud and Rie cautiously advanced the opinion that it is probable the number of cases in which epilepsy develops is slighter even than statistics indicate, and it is barely possible that some cases of epilepsy which develop many years after the occurrence of these palsies result from some other causation.

The symptom which is most distressing of all and which attends these cases with deplorable frequency is that of limitation of mental development. This varies in intensity from slight diminution of powers of reasoning and of moral sense up to complete idiocy. Generally, the mental symptoms show themselves soon after the occurrence of an attack, that is, in cases in which the paralysis does not occur before the period of development of any mental processes. Generally the child is late in learning to talk; in fact, in many cases it does not indulge in the echolalia which is the precursor of rational and purposive speech in a child. Sometimes they learn a word or two, but even these they fail to use with any degree of intelligence. If the attack occurs after the child has learned to talk and to display other intellectual accomplishments, there occur, in a large number of the cases, aphasic speech disturbances commonly of the motor kind. In a few cases there have been found some of the simpler forms of sensory aphasia, such as word-blindness and word-deafness, but these, compared with the frequency of motor aphasia, are very rare. Many of these children become the greatest burden imaginable to their parents and attendants, not alone because of their feeble-mindedness, but on account of their incessant activity. Never for a moment are they quiet in voice or in limb except when asleep. Many of them are vicious, destructive, cruel, and entirely unamenable to any form of pedagogic influence. Frequently some of their perceptive and receptive centres may be comparatively well developed, and it has gone abroad that some of them even may be talented in certain narrowly limited directions. It is, however, the discrepancy which exists between their general mental make-up and inordinate development of some mental faculty that gives the semblance of truth to this opinion. Although these cases are not the kind that furnish mathematical and musical geniuses, as some forms of idiocy do, occasionally it is noted that they are fond of music; but this should be taken to indicate that they are more capable of being distracted somewhat by sounds than in any other way, while others may be thus susceptible to other special sense stimulation such as through the smell or through

the sight. In most cases, however, the special senses are all obtunded. Objects brought into the field of vision, of hearing, of smell, or of touch interest them but momentarily, if at all. Many of the patients are filthy in their habits, and they do not differentiate between clean and unclean things, even with the discrimination of lower animals. On the other hand, some intellectual traits and capacities may be developed in them by education, but the higher qualities of intellectuality, such as respect, affection, moral sense, always remain submerged. In those cases in which the mental defects are not sufficiently marked to be considered a form of idiocy, there will be such evidences of mental shortcomings as forgetfulness, peevishness, irritability, loss of power of concentration and association, and particularly loss of inhibitory control of the appetites and passions. The truth of the latter is manifest in the popular saying: "Beware of the wrath of the cripple."

The sensory sphere is not liable to any special perturbations in cases of infantile cerebral palsy. In a few cases there have been found defect of vision, manifested by hemianopsia or complete loss of vision, partial or complete deafness, and limitation of the other special senses. It is difficult in some cases to tell to what degree these defects are determined by the general mental deficiency. There is no reason, however, why they should not develop on purely anatomical grounds; although the destructive processes in cases of infantile cerebral palsy are most frequently located in the precentral and frontal regions, they may involve such portions of the brain as the occipital and temporal lobes, which are positively known to be special-sense centres.

The general appearance of a patient with infantile cerebral palsy is always a striking one (Fig. 15). It varies, of course, with the intensity and duration of the disease, with the form of the clinical symptoms, and especially with the patient's intellectual possessions. In some cases the head is very small, in others it is strikingly large, while in others yet it is decidedly unsymmetrical, the depressions of the skull often corresponding to the diseased part of the brain. If the psychical shortcomings are pronounced, they will be conspicuously manifest in the child's appearance. In a few cases there are pupillary anomalies, excessively contracted or dilated pupils, sometimes a considerable degree of paralytic or intention nystagmus, and very rarely evidence of oculomotor nerve palsy on one or both sides. Menz has mentioned the case of a child who had bilateral oculomotor palsy in connection with the typical picture of infantile cerebral palsy. The lesion was supposed to be in the left crura cerebri and passing over the middle line to involve the right motor oculi. As a rule the inner

branches of the third nerve are involved. In about fifteen per cent. of the cases some form of strabismus occurs, especially in the post-natal palsies.

The participation of the cranial nerves has recently been studied

FIG. 15.—A Typical Case of Infantile Cerebral Palsy.

carefully in 72 cases by König. In 12 cases the seventh and twelfth nerves, the two most commonly involved in infantile cerebral palsies, were normal. The seventh was normal and the condition of the twelfth was questionable in 8 cases. The seventh and twelfth were

both questionable in 15 cases. The twelfth was paretic in 4 cases, the seventh in 3 cases, and the seventh and twelfth in 18 cases. His statistics show that affection of the frontal and ocular branches of the facial is rare, and that paralysis of both of these branches seldom, if ever, occurs. He confirms the observation of Freud and Rie that mimetic facial movements occur with extraordinary frequency compared with hemiplegia of adults. In 36 cases in which the facial was affected, in no less than 30 cases could loss of mimetic power be made out. In some cases involvement of the facial was to be detected only by loss of mimetic movements. In only a few cases was the musculature of the seventh nerve in a spastic condition.

Involvement of the sensory trigeminus is of exceedingly rare occurrence; that of the motor trigeminus less so. Slight paresis of the masticatory muscles has been noted by König, and by Moeli and Marinesco. Optic atrophy may occur with any form of infantile cerebral palsy, and it is more often bilateral than unilateral.

The statement of König relative to the frequency of optic-nerve involvement must be accepted with becoming hesitation, as many physicians who have investigated this point very closely are at variance with him. Thus Sachs says that in upwards of two hundred and twenty-five cases he has not seen optic-nerve atrophy more than three times. The bilateralism and symmetry of development of the atrophy in König's cases lead me to suspect that he has included some cases of basilar syphilis.

In many of the cases a difference in the innervation of the two sides of the face on emotional display will be seen, such, for instance, as when the patient begins to cry, or on smiling. The spastic rigidity and paralysis, the hemichoreic movements and athetosis, all of which are generally associated with a profound fixity and deformity, go to make up a striking physical appearance.

In the clinical description above given I have constantly kept in mind the class of infantile cerebral palsies that occurs after birth. It therefore becomes necessary to mention any individual characteristics which those cases occurring before and during birth may have and which are not included in this description. Cases of congenital infantile cerebral palsy are supposedly associated with some perversion or defect of development, the anatomical basis being a degree of agenesis corticalis, porencephalia, or destruction due to vascular changes. These may be unilateral or bilateral, and corresponding will be the symptoms. As a matter of experience the bilateral forms are as common as the unilateral, if not more so; and in this they differ from the palsies occurring after birth, the majority of which are unilateral. The paralysis in these cases, particularly

when it is bilateral, is not apt to be so complete as the unilateral form. The condition known as double athetosis is likewise dependent upon cerebral atrophy, consequent on arrest of development or pathological conditions attending birth. In these cases of double athetosis all the muscles are in a state of contraction or semi-contraction, and the extremities are in more or less constant rhythmical movements which are seemingly exaggerated by voluntary effort. In striking contrast to the condition of the muscles of the extremity is the face. This remains relaxed and expressionless. A condition very analogous to that of double athetosis has been described by Freud under the title of choreiform paresis, or hemiparesis in which the paresis was very slight and the choreiform movements were very pronounced, and sometimes the choreic and athetoid movements occur together in such a combination that the state can only be designated by the name of athetoid chorea. These motor phenomena are evidently all dependent on similar pathological lesions, the individual forms being determined by the seat and severity of the lesions. In some text-books a form of infantile cerebral palsy, prenatal in the time of origin, occurring principally in children born before full term, and characterized especially by diplegia of a spasmodic type, particularly manifest in the lower extremities where it constitutes paraplegic contracture, has received the name of Little's disease, on account of this form of birth palsies having first been described by that author. This designation and that of spasmodic tabes, by which it is sometimes known, are gradually disappearing from medical literature, and the symptom complex is being considered where it rightfully belongs, in the category of developmental defects, although German and French authors still use the title of Little's disease. The salient points about this form of congenital spastic rigidity are that

FIG. 16.—A Case of So-called "Little's" Disease.

the rigidity which dates from birth may be present in all four extremities, but is most marked in the lower from which it does not usually ever completely disappear (Fig. 16). Its occurrence is not preceded by the convulsive, febrile, and other evolutionary phenomena of the ordinary forms. The reflexes are all exaggerated, ankle clonus is generally very intense and occurs spontaneously whenever the feet happen to get in position of extension. Paralysis, in the strict sense of the word, of any of the members is never present, and the disturbances of motivity in the upper extremity tend to diminish spontaneously and perhaps completely disappear; but the spasticity and contractures of the lower extremities are persistent and give rise to a striking deformity. In some cases of this disease the spastic condition is first noticed when the child begins to walk. This fact is oftentimes of service in distinguishing it from the spastic condition that develops with hydrocephalus, particularly in those cases in which there is but slight enlargement of the head.

The familiary form of infantile cerebral palsy develops as a rule, to which, however, there would seem to be most important exceptions, during the first year of life, and is characterized by a gradual cessation of mental development and a concomitant stunting of somatic growth associated with spastic or flaccid paralysis of the upper and lower extremities. Eclamptic seizures do not occur in this form. Optic atrophy leading to complete blindness, deafness, and nystagmus are common attendant features.

Course, Duration, and Prognosis.

The course of the infantile cerebral palsies is essentially a progressive one, the prognosis as regards recovery is unfavorable, and the duration may extend over that of a natural lifetime. In a certain proportion of cases occurring after birth, and which are variously diagnosed as brain fever and cerebral hemorrhage, death foreshadowed by fever, status epilepticus, or exhaustion follows soon after the occurrence of the symptoms. The great majority of cases, however, whether they be dependent upon causes occurring before, during, or after birth, become chronic. The cases in which there is the greatest chance of spontaneous recovery are those which occur in the early years of childhood with prodromal and beginning symptoms which suggest an acute inflammatory process, and in which epilepsy of a severe type does not develop. The degree of spontaneous recovery that will result in these cases is shown within at least two years after the occurrence of the disease. Cases that so far undergo restitution as to show no mental or somatic evidences of the disease after

this time are extremely rare. At least one-third of the cases develop such a severe form of epilepsy that it incapacitates them completely from in any way caring for themselves, and it is probable that at least two-thirds of all cases of infantile cerebral palsy become eventually a burden to their families or to the community.

The duration of the disease, that is, the pathological results of the disease, may extend over the natural span of life. Lannois and Pauly have studied the anatomical conditions found in a case fifty years after the onset of the disease.

The prognosis for partial recovery and for the avoidance of epilepsy and imbecility is better in those cases in which the paralysis, athetosis, or choreiform movements are of moderate severity and limited to one side of the body. It is extremely unfavorable, looking to any degree of spontaneous recovery, where symptoms of double athetosis, diplegia, or spasmodic paraplegia occur. I have now under observation a child nine years old who had cerebral palsy with prodromal and attending symptoms pointing to an acute inflammatory process in the brain, and in whom paralysis of one side of the body and weakness of the opposite leg developed, who has learned to walk, and who can use the side which was formerly paralyzed nearly as well as the healthy side. At the present time a moderate degree of imbecility, or, I might better say, an unamenability to pedagogic influence, is apparently the only residue of her former disease. She has never developed epilepsy.

In some cases the advent of puberty seems to have a beneficent influence on the moral and intellectual growth; on the other hand, it is often noticed that this epoch of maturity is delayed and when it does occur there is no corresponding unfolding of the mind. It does not affect the frequency of epileptic attacks as do sometimes the onset and recurrence of the menstrual epoch.

Morbid Anatomy and Pathology.

The morbid changes found in the central nervous system after death in cases of infantile cerebral palsies are characterized by their variability and dissimilarity. They are found almost exclusively in the brain, but, as we shall have occasion to point out, in some cases changes in the cord are also to be determined. When discussing the subject clinically, we found that the symptoms grouped themselves naturally according to the time of the occurrence of the disease into prenatal, natal, and postnatal; and when we come to study the morbid anatomy we find that the diseased conditions differ according to the temporal appearance of the affection. In the prenatal cases

the common defect in the brain after death is either that of porencephalia or the condition known as agenesis corticalis, a state in which there is apparently a simple lack of development in the pyramidal cells and the other cells of the brain and their constituents, or, we might better say, in the motor, sensory, and associate neurons which are within the brain, particularly the former. In the cases that occur at the time of birth, the common morbid conditions are those of meningeal hemorrhages and their consequences and the acute infections which may occur at the time of birth. In the cases occurring after birth, the postnatal ones, the anatomical lesions are those dependent upon polioencephalitis, whether it be acute or hemorrhagic, upon meningeal and subpial hemorrhage, and upon the various vascular conditions leading to apoplexy and its degenerative sequences.

It matters not what the pathological lesions are in the beginning of infantile cerebral palsies, they all lead with considerable uniformity to one condition of the brain, namely, atrophy. Atrophy of a small or of a considerable area of the brain, or indeed of an entire hemisphere, is the result of the inflammatory, sclerotic, hemorrhagic, degenerative, or cystic changes which constitute the active pathological lesions in these palsies.

Certain portions of the brain are prone to be the seat of these lesions. These are the motor area of the cortex, the frontal region of the brain, the basal ganglia, and, very rarely, the occipital and temporal portions.

In about one-third of all the cases of infantile cerebral palsy there is porencephalia of some degree. The cavity formations may be very slight, or they may be so great as to usurp a large part of the substance of the hemisphere, and communicate with the lateral ventricles. The next most common pathological condition is what may be called an interstitial sclerosis, that is, an increase of connective tissue, and a consequent atrophy of the inherent cells of the part, which causes a condition called atrophy with sclerosis. In very exceptional cases, these sclerosed areas are small and multiple. It may be that symptoms such as nystagmus and intention tremor, which are sometimes a part of the clinical picture so as to simulate multiple sclerosis, are associated with this pathological condition. In some cases, say from five to ten per cent., there is found a condition of simple atrophy of the cell structures of the brain, a condition that suggests that the proton of the part was properly formed and that development had begun, but that it had ceased before any considerable degree had been reached. In from about ten to fifteen per cent. of the cases the atrophy is the result of embolism, thrombosis, or rupture, and consequent softening,

cystic formations, sclerotic repair, and the like. In a lesser percentage of cases, particularly in cases dating from birth, evidences of meningeal hemorrhage will be found. Meningeal hemorrhage either quickly terminates the life of the patient or its residuent remains for a long time, so that when the case comes to autopsy there may be scarcely sufficient evidence of a previous hemorrhage to indicate its existence. Atrophy of convolutions, meningeal thickenings, and pigmentation may be the only evidence, and they are attributable to other causes. In a few cases the gross and microscopic examination of the brain reveals the presence or remains of a meningoencephalitis, that is, a thickening and sclerosis of the meninges with the commonly attendant vascular changes, adhesion of the meninges to a more or less extensive area of cortex, and a chronic degenerative atrophic process in the implicated cortex. Statistics show that meningoencephalitis is an extremely rare cause of infantile cerebral palsies. Rarer still is hydrocephalus, although one or two cases attributed to either the tuberculous or congenital forms of this condition is to be found in the statistics of almost every author who has analyzed any considerable number of cases.

There is no unanimity of opinion as to the source and antecedents of all the morbid conditions upon which infantile cerebral palsies may be dependent. The mistake has been made of endeavoring to group the pathological conditions all under one or two heads. The truth is that infantile cerebral palsy may be due to any hemorrhagic, inflammatory, or degenerative conditions that affect the cortical motor areas, the motor tracts, or the sub-stations in which such tracts are interrupted. The antecedents of these three pathological conditions comprise practically almost every deviation from normal recognized by pathologists. As has been said, the great proportion of prenatal cases are dependent upon developmental shortcomings and upon defects acquired during the intra-uterine growth of the child. Concerning the former, we know absolutely nothing, and our attitude towards the latter is not much more satisfactory. We may advance the hypothesis that porencephalia is a terminal condition of previous rupture or blocking of a vessel or number of vessels, but we cannot bring forward absolute proofs. We do know, however, that rupture and occlusion of cerebral vessels, due to degeneration of the coats of the blood-vessels, to thrombosis, and to embolism, do occur. We know, further, that such degenerations may be to all intents and purposes idiopathic, and we believe that they may be traumatic. There is considerable difference of opinion as to the original seat and form of the vascular lesion. Sachs believes that hemorrhages are the most common expression, while

Gowers argues that thrombosis of the superficial veins is often the initial lesion. All writers are agreed that birth palsies which are attributable to dystocia are most commonly dependent upon some form of meningeal hemorrhage which generally involves eventually, if it does not in the beginning, the subpial space and the cortex. This was originally demonstrated by Dr. Sarah McNutt, and her findings have been corroborated by others. This form of hemorrhage is usually described as intra-arachnoid or hemorrhage upon the arachnoid. On examination after death, thick, more or less diffuse blood clots are found on the surface of the arachno-pia. The severity of the hemorrhage seems to be expended on the posterior pole of the brain. If the pial space is not also the seat of the hemorrhage, it will usually be found to contain a quantity of sanious fluid. This is the form of hemorrhage that is found in infants whose birth has been long and tedious or aided by instruments, and who come into the world still-born or asphyxiated. In some cases it occurs in infants born in natural labor, while its occurrence in others has been attributed to the encephalic stasis resulting from twists of the cord about the neck. It has been said previously that most of the cases of this form of meningeal hemorrhage are dead-born, or die a few hours after birth, never having recovered from the asphyxiated or still-born state. Postnatal birth palsies are dependent upon a greater number of pathological conditions than are either of the other two, because in these the different forms of infections of the brain and possibly even of the meninges may be causative. There is, however, nothing peculiar about those pathological conditions which should call for separate description in this place. The fact that the lesions are often located in different and remote parts of the brain, as well as occasionally in the spinal cord, would indicate that the original trouble is oftenest in the vascular system. In one of the cases cited by Wallenberg, there was a hemorrhage in the right optic thalamus and corpus striatum, and in another a terminal hemorrhagic condition involving the anterior quadrigeminal bodies and the crura of the brain. Quite a number of cases of similar lesions are to be found in the literature. In cases in which there have been lesions both in the brain and the spinal cord, it is probable that the original condition was multiple emboli. Very rarely the primary lesion of infantile cerebral palsy is a tumor involving the motor area. Donkin has reported a case dependent upon tuberculous tumor of this region, and Dercum one of angiolithic sarcoma of the same region. Naturally, any of these lesions involving the motor tract will produce descending degenerations, which may frequently be traced into the spinal cord and upon which the spastic phenomena are dependent.

Diagnosis and Differential Diagnosis.

The diagnosis of cerebral birth palsies usually offers no difficulty. The complex of symptoms of spastic paralysis in the shape of hemiplegia, diplegia, or paraplegia, attended with the usual accompaniments of such palsies, the athetoid and choreic movements, the attacks of epilepsy, and the striking mental shortcomings make up a clinical picture that is quite unmistakable. There are four conditions, however, which it is always necessary to exclude, and this cannot readily be done in some cases in which such symptoms as epilepsy and mental shortcomings are not present. These are: (1) anterior poliomyelitis; (2) idiopathic epilepsy; (3) subacute Sydenham's chorea; and (4) tumor of the brain. The first-named can be excluded with safety if it be borne in mind that it is characterized after the acute manifestations have ceased by flaccid paralysis, by progressive atrophy in the involved extremity, by absence of myotatic irritability, and by reaction of degeneration to the electric current. It can only be confounded with those cases of cerebral palsy in which the paralysis is of the paraplegic type, very rarely when it is of the hemiplegic type. It is never accompanied by choreic movements, athetosis, facial palsy, epilepsy, or mental shortcomings, except coincidentally, and coincidences of this kind do not occur often enough to merit mention.

Careful physical examination will differentiate it with almost unerring certainty from cases of idiopathic epilepsy. In the latter disease there are lacking paralysis and evidences of secondary degeneration in the pyramidal tracts, athetosis and choreiform movements, and all focal manifestations. The only cases of epilepsy that cannot be excluded are those really dependent upon an ancient lesion of infantile cerebral palsy which has so far undergone restitution as to rob it of all motorial, paralytic, and irritation accompaniments, except the periodic explosions attending the epilepsy. In these cases the only use of a differential diagnosis is the satisfaction that it may give him who makes it.

The differential diagnosis from Sydenham's chorea which has assumed a chronic course and which is associated, as many of these cases are, with amyosthenia and mental sluggishness offers no difficulty except in rare instances in which the leading symptom of the cerebral palsy is some form of choreiform movements. In such cases, however, the detection of any of the somatic accompaniments of infantile cerebral palsy will put the case in the right category. Double athetosis of infantile cerebral palsy may have a considerable

resemblance to this condition, but the brusk, paradoxical, extensive movements of chorea, which are not commonly of the face but always of the extremities, can be easily distinguished from the slow rhythmical coördinate movements of double athetosis. In the former disease there is absolutely no contracture. In the latter there frequently is.

The conditions which have been described by Freud under the name of choreic hemiparesis will probably eventually be found to have the same morbid anatomy as chronic chorea. The differential diagnosis from tumors of the brain involving the motor cortical area does not give rise to very much difficulty, because the symptom complex of brain tumor, which includes headache, vomiting, and optic neuritis, is not a part of infantile cerebral palsy. When the differential diagnosis between these two conditions cannot be made, and when the suspicion of brain tumor is well founded, as a last resort an exploratory trepanation is justifiable—all the more so, as the form of infantile cerebral palsy in which the intervention of the surgeon is justifiable is the very one with which brain tumor is confounded.

Treatment.

The physician can accomplish as little in the treatment of infantile cerebral palsies by the administration of drugs and the use of other therapeutic procedures as in any disease which he is called upon to treat. Yet in spite of this we cannot dismiss the subject of treatment without considerable discussion. Parents as well as physicians appreciate the hopelessness of the outcome of the great mass of this class of diseases, and anxiously seek the trial of any experiment or procedure which holds out the slightest hope for relief. Insomuch as discussion of the efficacy of operation on the brain has during the past few years been wellnigh universal, it is necessary to refer to its utility in the infantile cerebral palsies of infants.

Cases of cerebral palsy of infants are rarely recognized until some considerable time after the occurrence of the disease. At least, this is absolutely true in the experience of the neurologist. They are seen by the general practitioner and by the obstetrician, but they are unrecognized as cerebral palsies in infancy and are considered forms of meningitis, brain fever, or palsies of peripheral origin. A number of them, indeed, are confounded with acute anterior poliomyelitis. Their diagnosis is usually first made when the palsy, the contracture, the choreic or athetoid movements, or the epilepsy and idiocy begin to dominate the existence of the child. This being so, the opportunity of using preventive measures has ceased to exist. This makes the

necessity of emphasizing the fact, that possibly one-third or even more of all the cases of infantile cerebral palsies could be prevented if the causes of dystocia and prolonged labor could be promptly removed, more urgent. The injuriousness of forceps delivery is slight when compared with the evil effects of tedious labor. The prognosis could often be materially influenced for the better if the cases dependent upon acute inflammation of the gray matter of the brain were recognized and treated at the beginning of the disease.

Cases that are seen during the acute manifestations and prodromal symptoms should be treated in the same way as one of acute leptomeningitis or acute encephalitis, particularly should measures be taken to cut short or cause abatement of the convulsive phenomena. This can best be done by the administration by mouth or by rectum of large doses of chloral and bromide, and the application of ice to the head. The immediate treatment of cases due to meningeal hemorrhage does not differ from that of ordinary cerebral apoplexy; absolute quiet is by far the most important measure.

The treatment of these cases when they come to the neurologist must be considered under the following heads: Treatment of the deformities, including the paralysis, the contracture, the athetosis, and the choreiform movements; (2) treatment of the epilepsy; and (3) treatment of the mental shortcomings. Much may be done to overcome the deformities resulting from the paralysis and contracture by the use of modern orthopædic appliances, and particularly if the progress of the deformity and contracture be at the same time opposed by the use of massage. Electricity in any form is not of the slightest service. Occasionally the surgeon may decide the necessity of single or multiple tenotomy before the application of orthopædic apparatus, and oftentimes this little operation is of considerable service in assisting to overcome deformity. Very little can be done either by medication or orthopædic appliances to counteract the athetosis, and very much less can be done to overcome the choreic movements, although in individual cases carefully constructed models in gutta-percha from casts of the arm or splints have been used with service in contributing to the patient's comfort; occasionally permanent benefit follows. In some cases of athetosis, the continual movements have become so aggravating and distressing that amputation of the part has been proposed and resorted to. In fact, I know of nothing more distressing than the cravings of a little patient eleven years old with athetosis now under observation, for amputation of the hand and wrist, which are in continual rhythmical movement and associated with pain-

ful spasms. But nothing is to be gained by resort to such heroic measures.

Treatment of the epilepsy is also most unfruitful. The usual dietetic and bromide treatment of these cases counts for but little. If the patient is brought thoroughly under the influence of the bromide and if the diet and bowels are carefully regulated, the number and severity of the fits may be somewhat mitigated, but nothing real is gained by such treatment, except that the existence of the unfortunate one is made more tolerable. Yet this is the treatment which the great number of cases must receive. Personally, after a considerable experience with the opium-and-bromide plan of treatment, I have come to the conclusion that more benefit is to be derived from this than from any other. It consists of the administration of opium in doses of from five to ten grains daily, according to the age of the child, and continued for a period of six weeks. At the end of this time the opium is stopped abruptly, and the patient put upon toxic doses of bromide, from a drachm to two drachms a day. After these large doses have been given for from two to four weeks, the dose is gradually diminished until evidences of the drug's toxicity disappear, and the patient is kept on this minimal quantity until the fits again begin to be of increased frequency and severity, when the opium treatment may be repeated. I have found that by repeating the opium course once or twice a year more can be done to hold the epilepsy in abeyance than by any other drug or combination of drugs; and that in the mean time pedagogic measures have greater effect and are more easily carried out. In fact, the orthopædic measures, the opium-bromide medication, and pedagogic efforts constitute the plan of treatment applicable to the great majority of cases.

The application of cranial surgery has a narrower field of usefulness in the brain palsies of infancy than in any other disease for the relief of which it has ever been suggested. During the past five years, and especially since the time when Horsley showed that the skull could be opened with greater impunity than ever before was dreamed of, and since Lannelongue claimed a considerable degree of efficaciousness for craniectomy in microcephalic idiocy, operation on the skull has been extensively employed, and more so in America than anywhere else, for the relief of epilepsy developing with the cerebral palsies of childhood. The results of these operations have been carefully weighed and found to be disappointing. The only cases in which the surgeon should be advised to operate are those in which the diagnosis of a focal cortical lesion can be made, and those in which symptoms denote the dependence of the symptom complex

upon meningeal hemorrhage or cicatrix. Even in these cases very little if any permanent benefit can be promised. Every case of epilepsy, it matters not what its origin may be, is temporarily benefited by operation on the skull, and in some cases it may be fitting to resort to operation for such a modicum of grateful relief. To sum up my individual experience with operation for epilepsy associated with the cerebral palsies of childhood, and which is based on a considerable number of cases, I may say that the opinion of Dana is also mine: It exercises none other than a pedagogic influence. The operation itself and its entailment of confinement, of careful nursing and attention, of regulation of the diet, all tend to influence these unfortunate patients for the better; but this influence is rarely permanent.

Treatment of the patient from the standpoint of the pedagogue is a most important one. The physician can do most good by giving this side of the treatment his careful attention, while the orthopædic surgeon looks out for deforming contractures. The unfortunate victims of infantile cerebral palsy and its sequelæ of mental defects and epilepsy are the pariahs of our public schools, and as a result any latent cultivatibility which they may possibly possess is lost. Parents should be urged to place such children early in schools, private and semi-public, which have been organized in many parts of this country, such as at Syracuse, N. Y., and Elwyn, Pa., where mental and physical discipline best adapted to these cases is administered according to the most advanced methods. Such methods of teaching and discipline are superior, in almost every case, to individual treatment by a physician and teacher, even in cases in which the financial side of the question has not to be considered, unless the teacher has had large experience with these patients.

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MULTIPLE SCLEROSIS.

Insular cerebrospinal sclerosis, disseminated sclerosis, multilocular sclerosis, *sclerose en plaques*, *Herdsklerose*, polynesic sclerosis, Charcot's disease, etc., is one of the commonest degenerative diseases of the nervous system. The truth of this has but recently dawned on the profession.

History.

The history of the disease can be told in a few words. About the middle of the first half of the nineteenth century the lesions of disseminated sclerosis were depicted in the plates of Cruveilhier's "Anatomie Pathologique" (1835–1842), and in Carswell's "Illustrations of the Elementary Forms of Disease" (1838). The disease interested these authors on account of what it resulted in, not because of its genesis and course. The disease was first considered clinically by Frerichs, of Breslau, in 1849. A few years later (1856) Valentiner made important anatomical observations of several of the cases which Frerichs had studied clinically. Previous to this (1855) Turck had given some perfunctory attention to it from a physiological standpoint. The study of the anatomical basis of the disease received its first impetus from Rokitansky and from Rindfleisch. The former inclined to the belief that the formation of the localized areas of sclerosis was the result of a previous inflammation of the brain substance. It was not, however, until some years later (1863) that an explanation of the histogenesis of multiple insular sclerosis, so plausible that it still has ardent advocates at the present day, was put forth by the celebrated pathologist Rindfleisch, of Zurich. He believed that changes begin in individual vessels and in their branches, similar to those described as chronic inflammation here or elsewhere; that these induce and are accompanied by atrophy of the surrounding nervous elements and a coincident formation of connective tissue, which constitutes the "islands." It will be seen further on how closely this statement of views resembles that held and advocated by Marie and some other recent writers who have during the past decade busied themselves with the pathology of this affection.

Although, as has been stated, Frerichs was the first to consider the disease clinically, and although the affection has been carefully described by Baerwinkel, who made note of the occurrence of tremor only on voluntary movement, by Zenker, and by Valentiner, it was not until Charcot, under the leadership of Vulpian, took up the subject

in 1862, that the impetus was given to the clinical study of the condition which soon secured its universal recognition. Four years later Charcot published a lecture on *sclerose en plaques* which to-day remains a mirror held up to the disease as it unfolds itself in its classic form. If the reputation for diagnostic perspicaciousness of the famous French neurologist rested on this lecture alone, it would be established. He studied the disease not only clinically, but anatomically, and made in the latter field an important observation, to wit, that even when a nerve became ensheathed with the newly formed connective tissue, all its constituents suffered impairment up to the point of complete destruction, except the axis cylinders. Like so many other important contributions made by this neurologist, corroboration of the occurrence of symptoms and their dependence on certain morbid states came from the labor of his pupils, which he inspired and carefully fostered. Thus it was that Ordenstien, in his thesis (1867), dwelt upon the distinctive features between multiple sclerosis and paralysis agitans; that Bourneville and Guerard (1869) emphasized the significance of nystagmus; and although Charcot was afterwards prompted by wider experience to modify his views as to the frequency of occurrence of intention tremor and nystagmus, they still remain important integers in the make-up of every typical case. Since that time the disease has received the most widespread and ardent attention from physicians the civilized world over. The names of the most important contemporaneous contributors will be found in the body of this article and in the bibliography appended.

Etiology.

Although many important etiological factors of disseminated sclerosis are established, the real causation of the disease is unknown. It is one of the degenerative diseases of early life, and may occur at a most tender age, an apparently well-developed case coming on before the second year having been reported by Dreschfeld. It usually develops before the age of complete adolescence; its primary manifestations may be said never to show themselves later than the thirty-fifth to the fortieth year. I feel convinced that those who teach the occurrence of the majority of cases between the third and fourth year (Moncorvo) are in error; at the same time, decided inclination is had to the opinion of Buzzard, Oppenheim, and others, that in a great number of cases the advent of symptoms antedates by years the period stated by patients. An extensive search through the literature of this disease has revealed no less than twenty-seven cases in which the disease began during childhood.

This will be more readily comprehensible when it has been shown, as I shall endeavor to do later, that in the large number of cases which do not conform to one classical type of the disease, the time intervening between the first symptom and the accompaniments necessary for the diagnosis of the disease is a variable and often a long one; and that the interim may be without complaint or manifestation.

The disease has gone down in literature, on account of the dictum of Charcot, as one that occurs more often in females than in males; but my experience, corroborated by that of others, is that sex is not an etiological factor, the disease being quite as likely to occur in one as in the other.

Though the disease has never manifested itself in such a way as to cause it to be considered strictly familiary or hereditary, some instances have been recorded in which it would seem heredity was a factor. Such are the cases reported by Erb, Dreschfeld, Leuch and Frerichs. Pelizaeus has also reported five cases developing in the same family. After a careful examination of the records of his cases, and in the light of what is to-day known of hereditary cerebellar ataxia, I am unwilling to believe that the cases reported by him belong in this category. That a marked inherited, neuropathic disposition exists in many patients who develop this disease is shown by a number of cases.

The factors to which the occurrence of multiple sclerosis can often be attributed, named in the order of their importance and frequency, are: previous infectious disease, such as pneumonia, influenza, scarlatina, measles, typhoid fever, diphtheria, variola, dysentery, malaria (Torti and Angelieni), cholera, and puerperal fever; slow poisoning by some of the metallic elements, such as lead, copper, mercury, zinc, phosphorus, and carbon, and thus indirectly traceable to occupations; exposure to cold, and particularly if associated with hardship and inclement weather; psychical shock and strain, such as fright, and it would seem that the latter is particularly operative if associated with some slight injury, grief, and such reverses and excesses as make a profound impression on the nervous centres; and, possibly, inherited and acquired syphilis.

The rôle played by infection in the causation of this disease is in many cases so important that it must be given the first place. I have seen two striking examples of the disease distinctly traceable to such causes, one in a young woman of twenty-three, in whom the first manifestations of the disease followed close on an attack of tropical dysentery when she was four years old; and one in a man of twenty-seven, after influenza. Its causal relationship to the other infectious diseases above enumerated has been dwelt upon by Kahler and Pick,

Marie, Oppenheim, Ribbert, Leyden, and many other investigators; while the number of writers who have recorded cases in which an infectious disease had preceded the first symptoms of multiple sclerosis by a short time is very great. How infectious agencies, diversified as they are, operate to produce a destruction of tissue in scattered areas which have no interdependency, with a simultaneous development of degenerated tissue, and the processes that mediate these states, is a riddle, and proffered explanation must be theory based on analogy. We have gone no further with blood pathology than to be able to say that it is probable that the products of low forms of vegetable life, as well as certain organic substances taken in from without and forming compounds with some of the constituents of the blood, are frequently inimical to the preservation of the nutritive balance of various parts and constituents of the nervous system. What determines the destruction of one part after one toxic condition of the blood or the occurrence of an inflammatory process in one instance, and a purely degenerative one in another following a similar infection, we are entirely unable to say. That the presence of divers specific organisms in the blood, as the result of divers specific diseases, may facilitate the activity of a vulgar pathogenic microbe, and that thus a combined infection causes exclusively sclerosis in plaques, as suggested by P. Marie, is fanciful to a degree.

Reference to those cases of multiple sclerosis which follow infectious diseases such as measles, scarlatina, and diphtheria, as false disseminated sclerosis, as has been done by Dawson Williams, Boinet and Salebert, for example, is not only misleading but positively antipathic to our conception of the pathology of the disease. There would seem to be little doubt that the cases reported by the first writer under this title really belong in the category of acute hemorrhagic encephalitis. If there is such a condition as false disseminated sclerosis, it is acutely toxic or hysterical, not post-infectious and organic.

Prolonged absorption of some of the metallic poisons above mentioned has recently been reverted to as a cause of this disease by Oppenheim. Only one case has come under my own observation in which one of these substances, zinc, could be regarded as an etiological factor. Records of cases which could be distinctly traced to exposure, fright, and slight trauma are numerous. In thirteen cases which I have had under personal observation, none of the above-mentioned factors except the second and slight injury has played any part. It does not seem to me praiseworthy to take too seriously those cases in which it has been reported that the disease was traceable to a fit of anger, such as one reported by Jordan. Certain it is

that the disease may show itself or first be noticed after such an outbreak, but to admit such coincidence as evidence would be tolerated in no other annals than those of medicine. Fright, exposure, and the like are often the immediate auspices under which the symptoms of multiple sclerosis are ushered in; but how far they can be considered strictly causative, it is difficult to say. No fact is better established in neurology than that these occurrences debilitate the nervous system, and thus facilitate the occurrence of nutritive changes which are called disease.

From time to time the importance of inherited and acquired syphilis in the etiology of this disease is urged by some observer. The most unswerving advocate of this view has been, and is, Moncorvo, of Rio Janeiro. He has recently written of twenty-one cases, occurring in patients of from sixteen months to seven years of age, in the majority of which inherited syphilis was believed to be the cause of the disease. Hereditary syphilis, he says, is often proved by the occurrence of pathognomonic symptoms that respond to treatment. He believes, furthermore, that inherited syphilis causes multiple sclerosis in such a way as to coincide with the pathological hypothesis of Marie and Jendrassik—that is, that a perivascular lesion is the starting-point of the various nodules of sclerosis. Individual experience and examination of the literature leads me to deny any particular relationship between either inherited or acquired syphilis in the causation of multiple sclerosis, although it may be possible to get a history of syphilis in a very small percentage of the cases. The experience of the last-named observer is unique, and until corroborated should not be given recognition.

Multiple sclerosis occurs at an age before enervating habits and injurious excesses are acquired or indulged in, and thus they do not enter into the etiology of this disease, as they do in that of many other degenerative diseases of the nervous system. Although Tweedy has reported a case in which not the slightest causative influence except masturbation could be traced, that this is other than a mere coincidence can scarcely be credited. Some writers would have us believe that refrigeration, exposure, getting wet through, and the like, play an important rôle in the causation of this disease. For instance, Krafft-Ebing states that out of one hundred cases forty were attributable directly to these factors; and, on the other hand, he does not think that infection plays a very important part. My experience does not coincide with his. Cramer has noted the occurrence of multiple sclerosis in connection with acute myelitis, and a number of authors, particularly Kelp, Schüle, Buchwald, Schultze, and Hess, have pointed out its coexistence with diffuse sclerosis.

Symptoms.

The clinical phenomena presented by this disease are not easy to portray, because of the many different forms in which it reveals itself. When we consider the various locations which the islets of sclerosis may have in the different parts of the nervous system, and to which they may be limited for at least some time, this is not at all surprising. A brief description of the disease, as it shows itself typically, will first be given; and then the atypical forms will be considered. The symptoms are sometimes divided, to correspond to the predominant location of the sclerotic patches, into symptoms referable to the spinal cord, to the oblongata, and to the brain. This is a division which does not particularly facilitate comprehension of the disease, while it does add to the difficulty of recognizing the individuality of the affection. That symptoms frequently indicate the involvement of one of these parts in the beginning is true; but for the lesion to remain confined to either the brain or the cord during the entire course of its disease, therefore causing corresponding symptoms, is of such rarity that it need not be considered. The early symptoms are not alike in all cases, but they usually indicate primary spinal-cord involvement, although cerebral symptoms may be the predominant ones for some time. In the one there will be a history of unsteadiness and weakness in the legs, coming on after some trivial accident, which the patient inclines to believe may be the cause of the weakness, but more probably the presence of the disease is the cause of the accident. Such an onset I have seen in a peddler, twenty-eight years old, who, trying to walk on a plank thrown across a pool of water, fell, but did not hurt himself. He changed his clothes quickly, and there was no bad result, such as chill, from the wetting. Three days afterwards the first symptoms of multiple sclerosis, consisting of a feeling as if all the strength had left the arms and hands, crawling sensation in the lower extremities, and a change in speech were observed, and the disease developed progressively up to a certain stage from that time. In some cases, particularly observed in females, the symptoms of the disease will be ushered in by a sudden loss of power in the limbs, localized numbness, pins-and-needles sensations, sometimes confined to one extremity and more often in the one in which there is motor weakness, associated, perhaps, with some loss of sight. These symptoms may all disappear, and after a time recur in another extremity. Occasionally, in addition to the stiffness of the legs, which is generally associated with a good deal of disturbance of locomotion, there will be some hurried action of the sphincters, and occasionally a pain in one

extremity or in both. This pain varies in character, and although it may be sharp, similar to that of locomotor ataxia, it is usually of a dull, aching character, and persistent, not intermittent.

The trouble in walking is a very variable quantity. At first there may be little more than a simple spastic gait, which is associated with some loss of strength; the latter is particularly apparent after prolonged muscular effort. As the disease progresses the spastic gait becomes more pronounced, and the unsteadiness and staggering in walking are more apparent. On endeavoring to walk, the heel comes off the ground rather quickly, and there is difficulty in lifting the toe; but when it does clear the ground the foot shoots ahead like a spring, and frequently gets in front of the other—that is, there may be what is called a modified cross-legged progression. This dissociation between the movement of the legs and that of the body is very striking. The legs progress first and seemingly more or less involuntarily, while the body follows later, as in a completely voluntary condition. This spasticity is most manifest in the lower extremities, although it is frequently seen to a considerably less extent in the upper extremities. With it, myotatic irritability is increased. The knee jerks are very lively, and ankle clonus is a common concomitant. The latter is frequently seen when the patient rests the foot on the toes. This puts the Achilles tendon on the stretch, and the consequence is a rapid vibration, which frequently extends to the entire body, and is stopped only after complete rest. Another manifestation of myotatic irritability, which can often be demonstrated by the physician, is seen if the foot be dorsiflected, when the tibialis anticus muscle assumes a state of tonic contraction, and will not infrequently remain in this condition for some time.

In some cases the gait is typically cerebellar; that is, titubation, unsteadiness, and reeling in the act of progression are very striking. In addition to the spasticity, the patient walks as if inebriated. But the most common gait in the early stages of this disease is a combination of the cerebellar gait and spasmodic—in addition to the characteristics of the gait which have previously been described, there will be added some uncertainty of station, titubation, etc. In some cases the motor disturbance will, almost from the beginning, assume the shape of a partial or complete paraplegia, in which the "clasp-knife" rigidity is very similar to that found in transverse myelitis. This condition is almost always seen in the lower extremities, but it may involve one of the upper. Such paraplegia may, according to the patient's statement, come on rather abruptly. Hemiplegia, with or without preceding or subsequent disturbances of consciousness, does occur, but is not very common. This symp-

tom will be commented on later when referring to the atypical forms of the disease.

Oftentimes the weakness and stiffness in both legs, coming on suddenly and associated or not with other symptoms which we consider pathognomonic of multiple sclerosis, increase, so that in from six to eight weeks the patient cannot walk. Then there may follow apparent recovery, later a relapse or the development of similar conditions in other parts of the body, and eventually, perhaps several years later, the development of a typical multiple sclerosis. The early symptoms are often referable to the general nervous system, or there may be such general cerebral symptoms as vertigo, headache, and mild mental symptoms. On the other hand, among the first symptoms may be those referable to the eyes. A case reported by Oppenheim showed disturbance of vision, confined to the left eye, which developed acutely some years before the spastic paresis of the leg, optic-nerve atrophy, disturbance of sensibility, and other symptoms indicated the true nature of the disease; while in still other cases the disease can develop under the mask of encephalitis pontis, and in these cases symptoms referable to disease of the individual structures of the pons, such as ocular muscular paralysis, may be the symptoms of the onset. Very rarely do the disease manifestations confine themselves to one side of the body, as do for a long time the symptoms of paralysis agitans, a disease with which multiple sclerosis is often confounded. It must be remarked, however, that the spastic manifestations may be limited to one side.

Of the important motor symptoms, which in a way must be considered pathognomonic of the disease, tremor or trembling of the hands and the other extremities of the body holds the first place. This tremor is of a coarse, slow, rhythmical character, three to five per second. It has been individualized as an intention tremor, that is, one that occurs unequally on movement, and ceases completely when the part is at rest. The test which is usually employed to show this and to distinguish it from the tremor of paralysis agitans is to ask the patient to take a glass of water in one hand and carry it to the lips. The hand approaches the glass with a good deal of steadiness and grasps it, but the moment the hand begins to approach the mouth the tremor increases and continues to become intensified until the glass is near the mouth, when it becomes so great that the water is spilled, or the glass is violently thrown from the hand, and the tremor then involves the whole upper part of the body, including the head. The tremor, however, does not limit itself to voluntary or purposeful movements, but occurs also in reflex movements; therefore the designation "intention" is wholly inadequate. But it is difficult

to find a term which expresses so much of the important characteristic of the tremor as does this, and therefore it is becoming to retain it. As has been said, the tremor is most commonly manifest in the hands; in some cases it shows itself unequally in the head and in the upper part of the body, and it is intimately associated with the necessary muscular activity which is required to preserve the upright attitude. In some cases the tremulous manifestations are more common on one side than on the other. Very rarely the tongue and lips and vocal cords show analogous movement to this tremor, and when they do it signifies that sclerotic patches have developed in the oblongata. The intention tremor of multiple sclerosis is increased by all forms of emotion and excitement, and by taking alcoholic liquors. It is diminished by anything that diminishes muscular activity, and completely ceases when the parts are compelled to rest. For instance, in a patient with most violent intention tremor of the cephalic extremity, the preservation of the balance of the head by the slightest support is followed by complete cessation of the rhythmical movements, which begin, however, with increasing severity when the muscles on the one side or the other are called into activity in order to maintain the equilibrium of the head.

The next most important motor troubles are those referable to the eyes. In fact nystagmus is considered the most significent symptom in leading to a diagnosis, but true nystagmus—that is, pendulum movements around the position of rest—is not found in a very great proportion of the cases that have been carefully observed and commented upon. The nystagmus and nystagmus-like twitching are almost always horizontal, rarely vertical, and in a considerable number of the patients the movements continue when the eyes are fixed directly ahead, and are readily demonstrated when the point of fixation is brought towards the temporal or nasal side. This nystagmus must be looked upon as an associated disorder dependent upon insufficient cortical innervation of the nuclei of the ocular muscles. The nystagmus is due to an interruption of conduction between the oculomotor cortex and the nuclei of the ocular muscles. Very rarely is the nystagmus unilateral, although one such case has been reported by Norris. The other motor defects found in the eye in this disease are not at all typical, and are inconstant compared with the nystagmus. In some cases there is found a distinct restriction of the mobility of the eye which may or may not be associated with nystagmus. In 100 cases of multiple sclerosis in which the motor defects of the eye were investigated by Uhthoff, there were 6 cases of abducens paralysis, which was unilateral in 4, 3 cases of pressure motor-oculi paralysis, 3 cases in which there was paralysis of convergence, and 2

cases of ophthalmoplegia externa, making thus 17 cases in all. In addition to these disturbances in the external muscular apparatus of the eye, in a few cases there are pupillary anomalies, of which myosis, stiff pupils, and increased reflex excitability of the pupil are the most common, but they are in no way characteristic of the disease. It has been estimated by Parinaud and others that pupillary changes are found in about fifteen per cent. of the cases.

Disturbances of speech are among the most prominent motor troubles of the disease. The change, moreover, is not confined entirely to word production, but is shown also in vocalization. The three most prominent characteristics of speech in this disease are that it becomes scanning or syllabic, that it is produced only by apparently great muscular effort, and that it is all pitched in the same key, a monotone. This production of speech by apparently powerful effort is sometimes very striking. The patient, on being asked a question, will seemingly wait and hesitate, the face will assume an appreciative expression, as if about to speak; that is, there will be all the premonitory phases: the brow wrinkles, the blood-vessels on the forehead stand out, the face assumes a more crimson hue, and at last, almost at the point seemingly of disaster, the words will come forth in an explosive manner, but the rhythm will be slow and scanning and drawling. In some cases there has been noted marked trembling of the vocal cords, particularly in the cases reported by Krewicky and by Collet. The trouble in speech, however, may be explained in a way analogous to that which has been given for the nystagmus, that is, there is an interruption of cortical impulses going to the innervation of the muscles which preside over sound and word formation.

Of the special senses the one that shows involvement more frequently than any or all of the others is that of the sense of sight. It has been said that visual disturbance may be the introductory symptom; it may now be said that some disturbance of vision is as characteristic of multiple sclerosis as any one symptom possibly can be. In the beginning the disturbance may consist of slight obscuration of visual acuteness limited to one eye, which may be transitory, or at least which may so completely disappear that the patient no longer has cognizance of it, or it may be bilateral, severe, and dependent upon profound optic atrophy. Buzzard has adduced evidence to show that some degree of optic atrophy was present in forty-three cases of multiple sclerosis out of one hundred. Uhthoff, who has studied the ocular manifestations with great care, differentiates four forms of visual troubles which may occur in this disease. In the first there is central scotoma, with conservation of the peripheral visual field.

In the second there is central scotoma with concomitant retraction of the peripheral visual field. In the third form there is peripheral contraction of the visual field, which is irregular, and a preservation of central vision or at least relative intactness of central vision, and lastly a regular concentric retraction very analogous to that found in hysteria. This latter condition, however, is extremely rare. By ophthalmoscopic examination the changes to be seen are most commonly an excessive paleness, blanching, and atrophy, particularly of the temporal halves of the optic nerves; papillitis and complete atrophy of an optic nerve is very rare, as are also manifestations of post-inflammatory atrophy. Some cases show amblyopia without recognizable change in the discs. The other special senses, taste, smell, and hearing, are not commonly affected. Hearing is sometimes involved, impairment of this function appearing under the semblance of Ménière's disease, in which auditory paræsthesia and progressive deafness go hand-in-hand; and like those cases in which vertigo is severe and persistent, patches of sclerotic tissue in the cerebellum are held responsible for the symptoms. In those cases in which the syndrome of Ménière's disease is present, it is supposed that the tissues around the organ of Corti have become sclerosed.

The sensory troubles are not so common or characteristic as are the motor troubles, yet in a considerable proportion of cases pain at the onset, which has already been mentioned, numbness, pins-and-needles sensations in the extremities, particularly if associated with the motor conditions already described, are common features of this disease. Freund's contention that sensory manifestations play a much more important rôle in the symptomatology of the disease than is indicated by the text-books is supported in a measure by my own experience. I cannot, however, go so far as to say that from eighty to ninety per cent. of all the cases show some disturbance of sensibility, as does the author just quoted. Of thirty-three cases which he subjected to careful examination for sensory shortcomings, in twenty-nine there was some anomaly of touch sense, pain sense, pressure sense, temperature sense, or muscle sense. Stereognostic sensations, and sensations of localization in general, were not so commonly disturbed. In the cases in which disturbance of sensibility has been manifest, those that have come to autopsy have shown, contrary to the opinion expressed by Rabbie, a very considerable encroachment upon the gray matter by foci or islets of disease.

Somewhat less frequent than sensory troubles are symptoms referable to the viscera, and among the most common of these is loss of proper and efficient control of the sphincters. In not a few cases from the very beginning there is hurried action of the

sphincters, and patients are unable to put off for any time first calls. With this there is afterwards well-marked incontinence and impotence. The occurrence of these, to be sure, will go hand-in-hand with the development of sclerotic patches in the dorso-lumbar cord. These symptoms have not heretofore been remarked, but as the disease is more carefully studied, and as it gradually gathers into its domain many cases formerly considered under the head of hysteria and ataxic paraplegia, sphincter and visceral symptoms become more and more a part of the disease. Occasionally cases present themselves in which gastric symptoms, or persistent tendency to hiccough or vomiting, will betray the deposition of a sclerotic patch about the origin of the phrenic and motor pneumogastric nerves. Other symptoms indicative of involvement of the oblongata, such as defective innervation of the muscles of mastication, difficulty in deglutition, tremor of the larynx, glycosuria, and polyuria, may point to distinct invasion of this part of the nervous system.

Trophic manifestations are very rare, at least until the disease is extremely advanced, when from long-continued incontinence of urine and inability to maintain cleanliness bedsores may develop on parts of the body subject to pressure. They have no part, however, in the symptomatology of the disease.

The mental condition of patients suffering from multiple sclerosis is one that demands careful and earnest attention. In no other organic disease of the general nervous system, with the exception of general paresis, do mental symptoms play such an important part as they do in the disease under consideration; and it is perhaps on account of the lack of recognition of this fact, particularly of the recurrence of early and what might otherwise seem trivial psychical symptoms, that we owe the diagnosis of so many cases of multiple sclerosis as hysteria, a fact which no one can doubt. In the beginning the mental symptoms may be those of slight exaltation or depression or a tendency to mental vacillation accompanied with alteration in temperament and morale. These may develop very early and be slight, or they may be of an intense degree. Occasionally, even before the disease has progressed to such a stage that it is recognizable, there may be periods of transitory confusion and delirium. These disappear and the mind will be in apparently the same condition as before, but it cannot be too strongly emphasized that a vacillating perverted psychical state often accompanies the development of this disease from the beginning. In many cases there would seem to be impairment of mental equilibrium and a complete inability to restrain the emotions. One of the very first symptoms of a patient, who has been for long under observation, was an uncontrollable manifestation of smiles

and laughter, even on the most sacred and pathetic occasions, not to speak of their occurrence at other inopportune times. For a long time this, and a monotonous voice and bitemporal paleness of the optic discs, were the only symptoms to point to multiple sclerosis. The diagnosis, however, was made, and careful watch of the patient was kept for years. This scrutiny was warranted by the development of a typical picture of multiple sclerosis. In some instances the facial manifestations of the depressed emotions, crying, sobbing, etc., occur without corresponding psychical states, but they are not nearly so frequent as are the attacks of spasmodic laughter and smiling.

To show that the triad of symptoms—intention tremor, nystagmus, and staccato speech—do not furnish the only pathognomonic symptoms of multiple sclerosis, the words of Buzzard may be quoted. He says: "I am certainly disposed to believe that the occurrence of paresis in one or more limbs, with spontaneous recovery, and recurrence of symptoms in the same or another part, together with amblyopia, accompanied or not by pallor of one or both optic discs, constitute a symptom group which should hold a place in the diagnosis of insular sclerosis not second to that at present occupied by tremor on voluntary movement and scanning articulation."

The evolution of this disease and the relativity of the symptoms are strikingly shown in two patients who have been in the City Hospital almost from the beginning of their troubles. One patient is a man, thirty-two years old, who has always been a free drinker. Eight or nine years before the beginning of the present illness he got infected with syphilis, had the common cutaneous secondaries, and was treated for a number of months. Four or five years later he began to complain of weakness of the legs, which gradually increased so that at the time of admittance he was almost helpless, the paraplegia being of a spastic nature. The sphincters were slightly affected and the knee jerks were exaggerated, and there was a slowness, a pronounced drawl in speech. Examination of this patient a year later showed true nystagmus, typical scanning speech, bitemporal paleness of the optic discs, exaggeration of knee jerks, and the presence of ankle clonus, the lower extremities being nearly useless and spastic. Mentally power of association and readiness of perception were diminished, the mental attitude being that of contentment. Intention tremor has never been present.

The second case is still more instructive because it shows how extremely necessary it is to exclude multiple sclerosis before a diagnosis of other disease is made. The patient, a woman thirty-six years old, has been in this country about ten years. Seven months after she arrived here she got malaria and was under treatment for fifteen

months, during which time she became emaciated and anæmic. After that she was moderately well for a year or two, except that she had an attack of diplopia which was treated in an ophthalmic hospital for about ten months. Since the trouble with her eyes she has never been well. The evolution of symptoms in her disease has been as follows: Weakness in right leg, headache, weakness in left leg, complaint about vision, paræsthesiæ, unsteadiness of gait, stiffness of legs, exaggeration of knee-jerks, ankle clonus on the left side, pronounced nystagmus, paleness of optic discs, and slowed, measured, almost typically scanning speech. In this case also intention tremor has not yet developed.

Atypical Forms.

If we were to classify under the atypical forms all those cases which do not conform to the description just given, it would be necessary to describe a large number of conditions, for the non-conforming types are as numerous as the typical, and perhaps more so. It is, however, only when the disease simulates rather closely some other degenerative disease of the nervous system, such as bulbar paralysis, amyotrophic lateral sclerosis, transverse myelitis, combined sclerosis or ataxic paraplegia, and cerebral apoplexy, the features of any of which it may assume; or when the symptoms, after well-recognized manifestations of the disease have appeared, suddenly cease and the disease becomes abortive, constituting a *form fruste* of the French writers, that we consider it as belonging to the atypical forms. Charcot described the non-conforming cases under four heads: Atypical forms which are abortive owing to the disappearance of the symptoms, usually clinical examples of spasmodic paraplegia; atypical abortive forms owing to early arrest of development of the disease, which are also usually of the nature of spasmodic paraplegia; forms atypical or anomalous on account of the supervention of anomalous or unusual symptoms. Under the latter he would place those cases in which symptoms of tabes, amyotrophic lateral sclerosis, hemiplegia, and the like appear. This classification is sufficiently comprehensive to include all the atypical cases, though I must add that I have never seen a case of this disease in which there was permanent arrest of the disease process.

The forms that develop under the mask of chronic transverse myelitis or spasmodic paraplegia are the most common of atypical cases. This form is illustrated by a case which has been under observation for some years. A man who had previously been healthy began to have weakness and stiffness of the lower extremities which increased until paraplegia was nearly complete. With this was asso-

ciated hurried action of the sphincters, perversion of sensibility in irregularly distributed areas of the lower extremities, but in no place amounting to anæsthesia or analgesia, increased myotatic irritability and slight clonus; no muscular atrophy. After several months the paraplegia and other symptoms became ameliorated and it seemed as if the patient would recover. A year later there were such suggestive symptoms of disseminated insular sclerosis as attacks of involuntary laughter and grinning, diminished vision due to atrophy of the temporal halves of the optic nerve, and a modification in speech and intonation, which latter, although not at all that which is considered typical of this disease, was more or less measured and cast in a monotone. The further course of the disease has shown it to be unmistakably one of multiple sclerosis.

Therefore every case of paraplegia in which the spastic element is marked or in which it predominates should be carefully investigated, and particularly the patient should be closely examined for symptoms of multiple sclerosis when the paraplegia is seemingly getting well. For although the severity of the paraplegia may mitigate, this may simply be the forerunner, and an advanced one, of multiple sclerosis.

The atypical forms in which symptoms pointing to involvement of the oblongata are prominent are considered in the chapter on diseases of the oblongata, while other atypical forms are taken up in some detail under differential diagnosis.

Morbid Anatomy and Pathology.

The morbid anatomy of multiple sclerosis varies somewhat with almost every case. The essential lesion consists of islets of connective tissue, which are found irregularly distributed throughout the entire central nervous system. In one instance these patches predominate in the cord; while in another case, the cerebrum or other part of the intracranial substances will be the chief seat of them, the spinal cord containing comparatively few. For a long time it was taught (Charcot, Leyden, Obersteiner) that the islets of sclerosis developed almost exclusively in the white matter. The untenability of this view has been exposed by many, recently by Taylor, who states that the white and gray matter have equal predilection. In the brain the islets may be adherent to the meninges; sometimes they may be seen through the translucent membranes, or they may first be seen on the surface of the gray matter of the convolutions when the meninges are stripped off. Like in the spinal cord, these patches are more numerous and of greater size in the white matter than in the gray, although oftentimes the white and gray matter are

affected indifferently. On cross-section of the hemispheres one frequently recognizes the increased resistance to the knife, as it cuts through several sclerotic foci. Of the patches in the brain some are as large as the thumb, while others are so small that they cannot be detected by the unaided eye. They are generally of a rounded, lenticular, more or less irregular shape, sharply differentiated from the surrounding tissue both by consistence and color, although the latter is less obvious when the patches are in the white matter. The irregularity in distribution of the sclerosed patches, without relation to nerve tracts or nerve structures, is highly suggestive that the islets are related to the distribution of the blood-vessels, although of course this cannot be proved. The recent contributions of Demange, Ribbert, and Déjerine are in support of this view. Section through the midbrain, basal ganglia, the pons, and the oblongata, in the two latter of which they are often of comparatively immense size, shows the grayish, slate-colored, or grayish-red patches, encroaching on or usurping whole areas or segments which are believed to be concerned with the performance of exquisitely individualized functions. In other cases the islets involve one or more of the cranial nerves; most often, by far, the second. The islets of sclerosis are in the optic nerve itself, rarely in the optic chiasma and tract, and occasionally in the primary optic ganglia. Implication of this nerve is not limited to its course within the brain substance, for the extracerebral roots may be involved as well. The olfactory nerve is less often affected. In the spinal cord the islets may develop at any height from the oblongata to the filum terminale. The cervical and lumbar enlargements are favorite seats. Irregularity of distribution is characteristic of the islets in the cord. At one level they may involve the anterior columns of white matter, at another the posterior, while at another any combination of either of these two with the lateral, or involvement of the lateral columns alone, even to complete encircling of the cord, may exist. In the illustrations of the spinal-cord distribution of the islets of connective tissue, made by Charcot, and which have lent themselves to so many monographs and text-books since that time, the patches are limited to the white matter. This often gives an erroneous impression, for although the islets often predominate in the latter, they are frequently found, particularly those of microscopical size, in the gray matter.

If a sclerotic patch and the surrounding nerve substance be observed with the microscope, it will be seen to be of the slaty grayish or gray-red appearance, already described. The patch itself, of a homogeneous appearance to the naked eye, is seen to be exquisitely differentiated from the surrounding nervous substance. When

stained, it will be seen that the axis cylinders, which pass like electric wires in insulators through this mass which surrounds them, are devoid of neurilemma, and that between them there is a proliferation of glia tissue, the amount of which differs in each case. Although there are no changes in the blood-vessels, either in their walls or their number, which are constant or characteristic, in a large number of cases, especially in those investigated before the disease had existed a long time, distinct vascular lesion, in the form of hyaline thickening of their walls, and perivascular changes have been found. In some cases there is partial or complete multiple thrombosis of minute vessels. In ancient cases the vessels are often seen to be dilated, gaping, and with diseased coats. In addition to this there will be seen, depending upon the age of the patch, more or less numerous granular and amyloid bodies, drops of myelin and fat, and degenerate fatty cells, particularly in the peripheral portion of a patch. These have possibly some relationship to the essential lesion of the disease and indicate that the morbid process is still active.

In some cases, also, there will be found increase of round cells, which in certain localities, such as on the floor of the fourth ventricle, constitutes a typical proliferation. Huber's examination of a case seemed to show that the disease consists essentially in a parenchymatous degeneration of the nervous tissue. He found, in recently affected spots especially, that there was widespread disappearance of the nerve fibres, without any greater changes in the interstitial substance than could be accounted for by the changes secondary to the disappearance of the fibres, so that the sclerotic patches must be regarded as due to the disappearance of nerve fibres, with consequent proliferation of the interstitial substance.

It is because of the intactness of the axis cylinders, even when the myelin sheaths have completely disappeared, as they always do in this disease, that Wallerian or secondary degeneration, starting from the location of the sclerotic patch and descending or ascending, depending upon the kind of nerve fibres the sclerotic area interrupts, and reaching to the end of those fibres, does not take place. It is to this percolation of the sclerotic patches by axis cylinders, which retain their own integrity at the expense of their sheaths, that the explanation of the instability of all movements, which is an integer in almost every symptom of the disease, is referred. Motor impulses originate and start normally along their usual pathways, but at various levels, where a part of these impulses must be conveyed along axis cylinders which are robbed of their myelin sheaths, the continuity and integrity of the impulse become impaired. In a similar way the spasticity and disturbance of gait may be explained

by postulating a dissociation of the normal control and inhibitory relationship existing between the cerebellar and cerebral cortex and the motor executive apparatus. It is particularly in the fact that the areas of disseminated sclerosis enclose intact axis cylinders that this form of sclerosis differs from the diffuse form. In the latter the degenerated tissue is homogeneous, in the sense that it contains only one form of tissue, viz., degenerated; and all the tissue has undergone or is undergoing necrosis. The term multilocular sclerosis is sometimes used to designate a form in which all the sclerosis is irregularly distributed and in patches, but very numerous and very irregular in form, and in these patches axis cylinders are often found partially or completely destroyed. Marie has referred to the severity of the physical symptoms and the gravity and profundity of the mental symptoms which patients with the latter condition have, and Babinski has proposed to call this "disseminated sclerosis of a destructive form."

The pathology and pathogenesis of the disease have received various interpretations. Charcot, Uhthoff, and others who have studied the disease clinically as well as anatomically, and Weigert, assume a primary disease of the neuroglia. Marie, Jendrassik, Ribbert, and others accept and advance the theory of Rindfleisch, who posited a primary disease of the blood-vessels. They believe that the infectious or peccant matter penetrates the circulation and injures directly the vascular system, and that the sclerotic process, beginning in the perivascular spaces, gradually implicates the surrounding tissue and causes atrophy and destruction of the nerve elements.

Adamkiewicz, as well as Schultze and Babinski, assume a primary affection of the nerve fibres, a primary parenchymatous disease possibly dependent upon or associated with disease of the small blood-vessels, which conduces to proliferation of the neuroglia and destruction of myelin. Although the general consensus of opinion is, perhaps, more in favor of endarterial inflammation and degeneration following infections, and therefore corroborative of Rindfleisch's theory, microscopical examination sometimes shows that the theory of a primary basis of disease in the blood-vessels does not hold good for all cases. This has been recently and convincingly proven by Taylor and by Redlich. The former shows that the sclerotic patches are not always found near vessels, that the vessels do not present any great changes in markedly degenerated areas, and in one case the vessels were found to be undiseased. It must be remembered, however, that the lesions in the case reported by the first-mentioned were very ancient. Other instances, such as one above mentioned by Huber, tend to corroborate the theory of Adamkiewicz, and at the same time show that the vascular changes are not the primary or the im-

portant ones. Thus it will be seen that something can be offered in support of each of these theories, and that each observer inclines to the one which his experience would seem to corroborate.

The theory which assumes a primary disease of the neuroglia, a primary parenchymatous disease of the nature of a chronic inflammation, which in turn is followed by a secondary involvement of the nerve fibres, seems the most improbable one and the least supported by the evidence at hand. The usual process of pathogenesis is probably the activity of the peccant material directly on the blood-vessels and through them on the nervous tissue, to cause primary degeneration, which in turn is followed by a variable extent of neuroglia change of a secondary nature.

In the discussion of the pathology of this disease we have followed the most universally accepted teachings. It becomes necessary, therefore, to refer to the claims—quite heretical, so radically opposed are they to those given—made by Popoff. This writer, who so far has advanced his views only in a preliminary communication, contends that there is no proliferation of connective tissue; the appearance of increased connective tissue is often given by the presence of the degeneration products which lie between the persisting nerve fibres; the neuroglia and its cells suffer destruction attended with granule formation, and the leucocytes that have wandered from the blood-vessels degenerate. The starting-point of the original process is to be sought for in the blood-vessel which lies in the centre of the affected area. The principal change in the vessel wall is cellular infiltration followed by thickening and concentric narrowing of the lumen. The first nerve structure to suffer is the medullary sheath, then the axis cylinder degenerates, and eventually the supporting tissue is destroyed. The most startling claim that the author makes is that the naked axis cylinders, which have been looked upon as constant features of the morbid anatomy of the disease, are not nerve fibres undergoing degeneration; on the contrary, they are nerve fibres in the process of regeneration. He believes that in the *locus* where new blood-vessels form there sprout from the end of nerve fibres new axis-cylinder processes, like those that occur after simple severance of a nerve, which goes on to regeneration, even to being clothed with a medullary sheath.

Course, Duration, and Prognosis.

The course of the disease, always chronic, is often an interrupted one; rarely is it uniformly progressive. Multiple insular sclerosis is in all probability the sequela of some disease, not a disease *sui generis*, and thus often the completed picture of the latter is far removed from

the beginning. Early symptoms having nothing characteristic about them are often not considered a part of the disease, and particularly so when they subside and leave the patient for a time untrammelled, which they almost always do. In fact, one of the most striking features of the disease is the interval that elapses between the first symptoms and the development of diagnostic phenomena; it may extend over months and even years. The halting progress of the disease is shown frequently by the motor affections, paresis and spasticity of the extremities, which in the beginning, after lasting for a time, will completely disappear only to be followed sooner or later by a recurrence of the symptoms in the same or another part. It is shown also by the fact that, although in many cases the disease can be traced to childhood, the diagnosis is not often made at that age, and this mainly for the reason that the symptoms then are very indefinite or that a period of cessation intervenes between the beginning of the disease and the time when it can be recognized. When the real diagnostic symptoms occur, they may be seemingly attributed to factors which it has been thought have some actual causative influence, such as fright, trivial injuries, exposure, etc., but which, if they are injurious at all, are only so by facilitating the progress of the disease and precipitating active symptoms.

In a very few cases there occurs a cessation of the symptoms and the activity of the disease seems to reach a standstill; more rarely yet does the disease terminate in apparent recovery. Charcot believed that this termination was not so uncommon as is taught, and he cited many examples to prove his contention, but other clinicians have not been so fortunate in their observations.

The duration of the disease is the longest of any of the degenerative diseases of the nervous system. Very rarely does it last less than ten years and often it lasts twice that long. Although the course of the disease is often characterized by long remissions and prolonged periods in which the disease is stationary, and even by periods of amelioration of existing symptoms, when the patient reaches a certain stage—a stage when the vegetative functions of the body begin to be derelict in the execution of their duties, when digestion is poor and absorption almost abolished, the development of anæmia and a cachectic state predisposing to acute infections such as tuberculosis, pneumonia, and the like—tenure of life is short.

The prognosis of the disease has been sufficiently considered in the above. The prognosis in regard to life would seem to be worse in those cases in which spinal-cord involvement predominates. This because of the direct avenues to infection and so to dissolution that are opened up by severe and persistent bladder and bowel trouble.

Differential Diagnosis.

The conditions with which multiple sclerosis may be confounded are first, most frequently and most easily, hysteria, its various forms and manifestations; second, paralysis agitans; and third, hereditary spinal and hereditary cerebellar ataxia. In certain rare instances it will have to be differentiated from some toxic tremors, particularly those of alcohol and mercury; from cerebral apoplexy, transverse myelitis, ataxic paraplegia, so-called syphilitic spinal paralysis, amyotrophic lateral sclerosis, and general paresis. The statement by Buzzard that of all organic diseases of the nervous system, disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria, will be agreed to by every one who has seen much of the former disease. Both of these diseases show themselves most frequently before or during early adult life, and the development of each may often be traced to some physical or psychical trauma. The occurrence of more or less motor paralysis, sensory disturbance, vesical symptoms, and tremor continuing for a time, then followed by amelioration or disappearance and later by the reappearance of similar symptoms in the same or other parts of the body, is common to both diseases.

Often the symptoms in the beginning point unequivocally to hysteria, but later developments show the case to be one of multiple sclerosis. A case related by Saundby is in illustration: A woman, twenty-three years old, had at the age of fourteen an attack of illness, in which the prominent symptoms were giddiness, tinnitus aurium, loss of the sense of taste, and weakness of the limbs. A year later there was added the occurrence of spastic paraplegia, which was followed by apparent recovery, all of which, it was thought, pointed to hysteria. Six years later there occurred such unmistakable symptoms of multiple sclerosis as ptosis, defective vision, pallor of the optic discs, nystagmus of an ataxic type, slow and hesitating speech, headache, vertigo, loss of tactile and muscular sense, intention tremor, paraplegia, exaggerated knee jerks and ankle clonus, Romberg's sign, and involuntary spasmodic movements of the lower extremities. A fact of great importance and one that should not be overlooked is that the two diseases, hysteria and multiple sclerosis, may exist simultaneously, and the former may not only mask the latter but may in some cases assume the mask of multiple sclerosis as well, so that the most experienced clinicians will be deceived. In support of this, instances reported by Westphal, Babinski, and others may be cited in which cases presenting a complete picture of multiple sclerosis were found to

be devoid of the lesion of that disease after death. I do not concede the necessity of a general neurosis, signified by the term pseudosclerosis, which Westphal would consider responsible for cases which do not correspond with the typical case of multiple sclerosis either in its course or symptomatology. They are, I believe, in some instances cases of hysteria and in others atypical forms of the disease under consideration, or possibly cases of brain syphilis such as was undoubtedly the case reported by Maguire under the title of pseudosclerosis and in which great improvement followed the administration of small doses of iodide of potassium. In this same category must be placed those cases in which symptoms indicative of multiple sclerosis yielded to the use of a drug such as solanin. It may be said without fear of contradiction, that if the cessation of symptoms were not merely coincident with the period of amelioration in an attack of multiple sclerosis, then the disease was hysteria, no matter how closely it simulated the former.

The occurrence of hysteria and multiple sclerosis together is in all probability responsible for the wonderful pathological conception put forth by some of the Charcot school that the disease hysteria, a mental one without known or conceived anatomical foundation, can occasionally in some way undergo a transformation the pathological result of which is a degeneration of nerve substance and neuroglia sclerosis, and thus eventuate in an organic disease. It can be taken for granted that hysteria is not one of the diseases that cause multiple sclerosis; on the other hand, that the latter may be the provocative agent of hysteria has been shown conclusively by Guinon. In a case that shows the symptoms of hysteria in the beginning and those of multiple sclerosis later on, it is much more reasonable to assume that the symptoms of the former are provoked by the organic disease than that transformation from hysteria to multiple sclerosis has taken place.

To diagnose hysteria from multiple sclerosis it is necessary to establish clearly the stigmata of the former. In this country it is uncommon to find hysteria of such severity that the classical stigmata will be easily made out, but disturbances of sensibility—either hemianæsthesia, total loss of sensibility of the entire half of the body, or a single area of hyperæsthesia—can almost always be determined by careful examination, and when found are important diagnostic factors, as such symptoms form a minor part in the make-up of multiple sclerosis and are probably never typically developed.

The paralysis accompanying hysteria is most often of the flaccid kind and the loss of power is usually complete, while in multiple sclerosis the paralysis is always spastic and the loss of power only

moderate. The contradictions of hysterical paralyses in their onset, course, and termination are often very striking.

Loss of sense of taste, of smell, and certain perversions of the sense of hearing, such as lack of perception of high notes on one side of the body, are valuable aids in the diagnosis of hysteria. Disturbances of vision are common to both disease, but in hysteria either concentric limitation of the visual field, achromatopsia, or complete amaurosis is the common condition, while in multiple sclerosis there is a partial obscuration of vision commensurate with changes in the optic nerve and which can be made out with the ophthalmoscope. Concentric limitation rarely occurs and color perception is never paradoxical. The significance of stigmata is therefore very great and it is by their presence that the diagnosis must be established.

The diagnosis of multiple sclerosis from hereditary spinal ataxia (Friedreich's disease) and hereditary cerebellar ataxia is often beset with great difficulties. In Friedreich's disease, however, there is gradual impairment of coördination, first in the legs, afterwards in the arms; the gait is more or less reeling; there is a quick backward and forward balancing movement; the involuntary movements of the head and extremities are twitching, irregular, choreic, not rhythmical; and the ataxia is not intentional. There is rarely paralysis of any of the eye muscles, and, most important of all, myotatic irritability is lost. These in addition to the fact that Friedreich's disease is often seen at an earlier age than multiple sclerosis, frequently in several members of a family, and is often accompanied by bodily deformities such as some form of clubfoot and spinal curvature, will facilitate the diagnosis.

Hereditary cerebellar ataxia may be still more difficult to differentiate from multiple sclerosis, and it may be quite impossible if the sclerotic patches confine themselves to the brain. In both diseases there may be the same titubating gait, and intention movements of the upper extremities, the same increase of myotatic irritability, nystagmus, and mental deficiency. Both of these diseases are often first discovered during youth, so that the most important differentiating help is the eliciting of a familiary or hereditary history in the former, a *sine qua non* of the disease. It is to be borne in mind that there have been reported cases of multiple sclerosis occurring in families, but if such occurrences are true examples of the latter disease they must be looked upon as accidental, for multiple sclerosis can in no wise be considered a family disease.

To differentiate it from paralysis agitans, a kind of time-honored demonstration, is most easy. The onset in late adult life with what

is termed rheumatic stiffness in one of the extremities, generally the upper; the occurrence of a fine tremor which persists during rest and which is not increased by voluntary, purposive, or reflex movement, at first confined to one upper extremity and very much later the other; the bent, stooping attitude; the characteristic immobile, masked face; the evidences of vasomotor perversion; flushings, perspiration, the preservation of mental integrity—are all a part of Parkinson's disease and correspondingly lacking in multiple sclerosis.

To discriminate between multiple sclerosis and the symptom complex produced by mercury and alcohol will not be difficult except in a few rare instances. The tremor resulting from chronic mercurial intoxication is very similar to that of multiple sclerosis in every particular except in its time of occurrence. The latter does not occur except on spontaneous or reflex movement, the former does. Moreover, mercurial toxæmia sufficient to cause tremor of long duration will be manifested by a cachexia which is very striking. Chronic alcoholism is frequently attended with tremor and nystagmus-like movements of the eyeballs, and these sometimes make it necessary to exclude multiple sclerosis. The character of the tremor, fine and rapid, its amelioration by all forms of stimulation, and aggravation by the withdrawal of stimulants, and the atypical twitching of the eyeballs are very suggestive. In chronic alcoholism there is always flaccidity of the muscles and myotatic irritability is diminished.

When apoplectiform attacks occur during the course of multiple sclerosis, as they sometimes do, it becomes necessary to distinguish it from genuine cerebral apoplexy due to hemorrhage or plugging of a blood-vessel. The most important factors in making this differentiation are: in multiple sclerosis the hemiplegia is never preceded by such premonitory symptoms as are often found to precede an attack of true cerebral apoplexy; during the hemiplegia occurring with multiple sclerosis the temperature is always elevated, while in true apoplexy it is first depressed and later slightly raised. The weightiest point in the diagnosis, however, is the transitoriness of the hemiplegic attack in multiple sclerosis and the absence of any secondary manifestations after it, such as evidences of secondary degeneration.

Multiple sclerosis may cause a typical picture of spastic spinal paralysis and may closely simulate the symptomatology of transverse and pressure myelitis. Lapinsky has mentioned a case under observation for two years in which the symptoms were typically those of spastic spinal paralysis, with absence of intention tremor and nystagmus, yet it was shown by the autopsy that the lesion consisted of islets of sclerosis in the brain and spinal cord. Generally, however, in these cases the limitation of the symptoms to the lower extremities

and the early involvement of the sphincters will suggest the diagnosis of spastic and spinal paralysis; and the same symptoms, with the addition of sensory irritation symptoms if the myelitis be due to pressure, will exclude multiple sclerosis.

On account of the comparative frequency with which some evidences of mental failure accompany multiple sclerosis, and on account of the fact that they have in common tremor, speech disturbance, amyosthenia, a stiff gait, and sometimes apoplectic attacks, it occasionally becomes necessary to differentiate it from general paresis. The history of preceding syphilis, the occurrence of a bizarre mental state before the period of exaltation, and delusions of wealth and grandeur; the peculiar thick speech with a rising inflection or intonation towards the end of a sentence, the continuous tremor which has no relation to voluntary movement, the profound mental troubles which are usually uniformly progressive, will early suggest and substantiate the diagnosis of general paresis. In cases which begin with symptoms indicative of brain involvement or in which such symptoms predominate, it is sometimes necessary to exclude brain tumor and encephalitis. This often cannot be done at once. As a rule such cardinal symptoms of brain tumor as vomiting, intense headache, and slowness of the pulse are absent in multiple sclerosis, and although the ophthalmoscope often reveals optic-nerve involvement in this disease, it is never of the nature of choked disc, and rarely is it apparently a mild form of neuritis which is quickly replaced by atrophy. When lesion of the disc revealable by the ophthalmoscope is present, it is paleness or atrophy of the disc, generally on the temporal side. It is from a consideration of these facts and from the different course of the two diseases, multiple sclerosis by progressions and remissions and brain tumor by gradual progression, that the diagnosis is to be made. Sclerotic patches in the cerebellum may simulate in the beginning neoplasm of that structure, but the absence of intracranial pressure symptoms will make the diagnosis.

It is barely possible that occasionally the necessity of differentiating this disease from chronic chorea and some forms of hysterical chorea may arise, but a careful consideration of the somatic accompaniments of the former disease will always suggest the diagnosis.

Ataxic paraplegia cannot be excluded with certainty in the beginning. I have seen cases of multiple sclerosis develop in adult life with symptoms which are considered diagnostic of the former disease, and while these cases were in the hospital under continuous observation, have seen added nystagmus, scanning staccato speech, and intention tremor—symptoms which point unmistakably to small multiple lesions in the conducting tissue of the central nervous sys-

tem. It is necessary, therefore, to make a provisional diagnosis in some of these cases, it being always borne in mind that multiple sclerosis is a possibility.

Treatment.

The treatment of multiple sclerosis may be summed up in two words—rest and nutrition. After the disease has developed, there is no medicament that has the slightest effect, either to influence the course or to thwart the progress of the disease. Measures directed towards securing bettered nutrition of the patient and as much rest as is consistent with bodily health, rarely fail to make the patient more comfortable and more tolerant of his infirmity. No one who sees much of hospital patients can fail to recognize the truth of this. Such patients by very reason of their stay in the hospital, with its *régime* and its discipline, often are more comfortable, and their disease seemingly makes slower progress than does that of private patients. It is not considered necessary to enumerate the plans by which such bettered nutrition may be obtained. At various times certain medicinal substances have been advocated, but none of them has stood the test of experience. It is probable that in those instances in which a certain drug, such as solanin, has been warmly recommended, its efficaciousness has been in cases of hysteria which have been mistaken for multiple sclerosis. If medicines must be administered it would seem most justifiable to give those substances which have already attained some remedial reputation in the degenerative diseases of the central nervous system, such as arsenic, nitrate of silver, etc. Indeed, the former was recommended by Eulenburg, and that hypodermatically. We have no doubt that in many instances drugs undeservingly have secured a reputation in this disease because their administration coincided or preceded a period of remission or temporary cessation of the symptoms, which are sometimes seen. As in all other organic diseases of the nervous system, electricity at one time or another has been advocated, but I have never seen the slightest benefit follow even its determined use. Patients who are at home in northern climates are much more comfortable in summer than in winter; it therefore follows that a more balmy climate should be their winter residence.

It is impossible yet to speak of the preventive treatment of this disease, except to reiterate that the medical profession is not aroused to a sufficient appreciation of the deleterious influences that the infectious diseases have on the entire nervous system.

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HYDROCEPHALUS.

Hydrops ventriculorum cerebri; Hydrocephalus internal, external, or intrameningeal; Water on the brain.

By hydrocephalus is meant, as the term signifies, a pathological increase of the intracranial fluid which may be either in ventricles, where the fluid is found naturally in largest quantity, or in the subarachnoid tissue, or in a space which it makes for itself.

History.

The disease is as old as the history and tradition of medicine. Less labor has been expended to solve the pathogenesis of hydrocephalus than of any other condition that occurs with half the frequency. The result is that as little is known of the disease to-day as was more than a half century ago—a statement that cannot be truthfully made of many diseases. In fact a careful survey of the literature reveals that the diaphanous description given by Watson served nearly all English writers up to the time of Hilton Fagge, who wrote in his inimitable way the best description of the pathology and symptomatology of this disease (although in the latter he improved but little if at all the former's description) that has ever been penned. In fact many so-called exhaustive descriptions of hydrocephalus since the date of Fagge's treatise have consisted of versions of this description, varying from those in which all his facts are put forth in new vesture up to those in which literal transcription masquerades as the reflection of personal knowledge; and of the latter there are many. In a similar way the description by Huguenin in "Ziemssen's Cyclopædia" has long represented the status of the subject amongst the Germans.

One result of this implicit acceptation on faith of incidences in

the disease both in its occurrence and in its symptomatology, in lieu of personal observation, has been that untenable contentions have been stated so often that they, like falsehoods industriously repeated, have seemed to become realities. For instance, in 1820 Bright gave the history of one James Cardinal, a patient in Guy's Hospital, as a case in which the subdural or arachnoid space was the seat of a large effusion and not associated with any brain malformation. And although it has been demonstrated beyond cavil that the fluid in the subdural space was the result of an escape of ventricular fluid through a hole in the corpus callosum, the case is still quoted as evidence of the occurrence of enormous intrameningeal hydrocephalus independent of malformation of the brain.

Much confusion has arisen, or at least students have had their perspective of this disease obscured by references to various clinical and pathological forms and subdivisions. I shall endeavor to avoid such confusion in this description. In the first place it needs be distinctly stated that the term hydrocephalus is used in two entirely different senses. In one, the sense in which it is used here, to express the occurrence of a pathological or disease entity, and the other to denote a symptom occurring in the course of a disease. As an example of the latter I may cite a case recently reported by Kretz in which death was caused by hydrocephalus resulting from an intrameningeal hemorrhage caused by an aneurysm of the internal carotid. Also a case which I have recently published of hydrocephalus arising from a tumor which completely occluded the aqueduct of Sylvius. This distinction should be clearly made. We no longer speak of acute hydrocephalus as a disease, we refer to it as a symptom of tuberculous meningitis or of that condition so lucidly described by Quincke attending meningitis serosa. No more should we dignify the occurrence of an increase of fluid with such conditions as porencephalia, atrophy of the brain, senility, depraved conditions of the blood, dropsy resulting from venous stasis, whether dependent on disease of the heart or other conditions preventing venous return, by a separate description or attempts to raise to an entity, than we should compensatory or consequential collection of fluid in other parts of the body such as abdominal dropsy. This form therefore finds no place in this description, but is relegated to the respective diseases or pathological conditions with which it occurs.

Etiology and Pathogenesis.

True hydrocephalus is genetically of two kinds, congenital and acquired. The acquired form is not infrequently secondary and

symptomatic. Anatomically hydrocephalus may be divided into internal, when the collection of fluid is predominantly within the ventricles, and external when exclusively in the subdural space. The latter is so very rare that it scarcely merits consideration. In the acquired form there is always an increase of fluid within the ventricles. That is, the acquired form may be accompanied by a subdural collection of fluid, but acquired external hydrocephalus only occurs symptomatically. The causes, the symptoms, and the pathogenesis of acquired internal hydrocephalus, are, so far as they are known, the same as those of the congenital form, the only difference being in the time of development. Therefore no attempt is made to describe them separately, nor can they be distinguished at the bedside. In order to emphasize what has been previously said it should be borne in mind that symptomatic hydrocephalus may be present with congenital conditions such as porencephaly. But such hydrocephalus is not considered congenital, although it may be symptomatic of a congenital condition. Clinically hydrocephalus is often spoken of as acute and chronic. Acute hydrocephalus is a symptom of some disease. Congenital hydrocephalus is always chronic.

Congenital hydrocephalus may manifest itself at any time after the fifth month of prenatal life or during the first months of post-uterine life, probably more frequently then; but this is difficult to estimate, for many of the cases that develop before birth are not taken into consideration in estimating relative frequency of occurrence, as they manifest themselves merely as one of the causes of dystocia and die in transit.

Very little is actually known of the causes of congenital hydrocephalus. Factors that have been found to precede it so frequently that they have been considered as causative are social transgressions of the parents, such as alcoholism, sexual excess, hyperfœtation, etc., and such physical disease and stigmata as syphilis, cachexia, and somatic evidences of degeneration. There is no dearth of evidence to show that consanguinary and familiary factors enter into the etiology, many writers testifying to the occurrence of congenital predisposition in more than one member of the family. Mental shock, and physical injury especially to the abdomen of the mother, seem sometimes to stand in causative relationship. The occurrence of infectious disease of the mother while she is carrying the child may also contribute to its occurrence. It is sometimes seen in infants who show other somatic defects such as harelip, cleft palate, spina bifida, and the like. Cases have been reported occurring with syringomyelia, but it is likely that these cases were examples of myelohydrosis, the distention of the central canal being secondary to the overdis-

tention of the ventricles, and especially is this the probable explanation of the cases reported by Rufenberg in which there were no symptoms of syringomyelia. In rare instances the hydrocephalus is partial, the distention being limited to one cavity. Such instances have been reported by Keen and by Robinson.

The pathogenesis of the disease is as obscure as the etiology. What the source and origin of the fluid within the ventricles is cannot be said truthfully. Most observers favor the view of Fagge, who taught that the most plausible theory to explain it was that which referred the excess of fluid to an inflammatory or possibly a degenerative process in the ependyma and choroid plexuses, and this he believed, furthermore, was the way in which many acquired cases develop. The facts that we have not yet definitely concluded what the functions of the choroid plexus are, whether it has any functions other than vascular, such as secretory, and that we are lacking in histological examinations of this plexus in cases of hydrocephalus, militate against the acceptance or rejection of this view. That there are other ways by which the hydrocephalus may develop is apparent from experience, which shows that in some cases mechanical obstruction to the flow of cerebrospinal fluid is the explanation of its development. Sometimes the obstruction will consist of an abnormal and imperforate medullary velum. It is very rare that such mechanical obstructions as closure of the foramen Magendie at the lower angle of the fourth ventricle or of the aqueduct of Sylvius, are alone accountable. They do occur, but as they are almost always the result of meningeal inflammation it is probable that the latter is as potent as the former.

In the acquired form, exclusive of the symptomatic varieties, the etiology and pathology are quite as obscure. In fact it is not at all improbable that the acquired form is in some cases identical with the congenital, only that it develops later in life. Such would seem to be true in the case of the noted James Cardinal, the case reported by Bondurant, and many others. Blows on the head, which do not apparently cause meningitis, and exposure to the rays of the sun are factors which are very often noted to precede the development of symptoms in acquired hydrocephalus. The forms that develop after infantile leptomeningitis as described by Huguenin, or after serous meningitis as described by Quincke, we look upon as symptomatic. In fact it is not hazarding one's prophetic reputation to say that hydrocephalus, like dropsy, is doomed to disappear as a clinical entity, and will soon be no longer entitled to any other description than that of a frequent symptom accompanying defective conditions before and after birth.

Simptoms.

The symptoms of hydrocephalus vary according to the time at which they begin to develop, and as to whether they are congenital or acquired. In the congenital form, if the hydrocephalus has attained some extent before birth, it will cause dystocia, and the life of a useless child is often sacrificed to spare the mother. If it is not so advanced as to cause dystocia or if it does not begin till some time after birth, the two striking symptoms attending its development are progressive enlargement of the head and absence of any trace of mental awakening. The circumference of a normal child's head shortly after birth varies from 35 to 40 cm. In hydrocephalus measurements every few days corroborate the information to be got by observation that the head is growing progressively larger; eventually it may become so large that it measures upward of 100 cm. in circumference. This enlargement is a uniform one in the sagittal diameter, and therefore the head becomes typically dolichocephalic. Some cases of congenital hydrocephalus without enlargement of the head have been reported, and these cases are to be looked upon as ones showing another sign of degeneracy, viz., premature ossification. Naturally the result of this enlargement is to cause first a bulging through the openings of the skull, if they have not been closed by premature ossification, or if they have, and later anyway, a separation of the bones of the skull at all parts except at their base and a thinning of the bones themselves. This separation of the cranial bones at their sutures and interference with the nutrition of the bones go on to such an extent that the fluid may be easily palpated through the thin and distended skull. In rare instances the fluid may break through and so discharge itself. This progressively increasing deformity gives the child a very typical appearance, particularly as the body suffers correspondingly from malnutrition. The weak, emaciated trunk and extremities with the small, puny, pinched features in contrast with the enormous head make a striking picture. If the hydrocephalus dates from birth and increases rapidly after birth the deformity will then be most striking, for the process of expansion is so much greater than the process of ossification that the ossified centres of the cranial bone are but as broken and matted clumps of ice on the surface of a pond in the beginning of spring. If the expansion of the skull has been a slower one and the nutrition of the skull bones not so completely interfered with, there will have developed in the angular spaces between the bones osseous formations known as ossa triquetra. In some instances these ossa triquetra are so numerous and so ad-

vanced in their formation that they close the head and militate against further enlargement except it be a very slow one.

As the child's head continues to increase in size the muscles that support and steady it suffer in nutrition, and these two factors combined, the size of the head and the weakness of its support, though the one were sufficient, prevent the head from being held up or from being supported or steadied; consequently when placed on a pillow it turns or falls in the direction of least resistance. When the hydrocephalus develops more slowly and later in childhood the enlargement of the head may be accompanied by increasing inability to maintain the erect position of the cephalic extremity, a shortcoming which the child endeavors to compensate for by supporting the face on either side with the palm of the hand or by letting the occiput sink back against the nape of the neck. The child in this position presents a strikingly characteristic appearance: the small pinched, undeveloped face; the vertically projecting forehead surmounting and overhanging the small eyes so as often to nearly obscure them; the bow-shaped increase of the temporal and parietal bones, so great that it nearly hides the ears; the projection backward of the occipital bone, this great enlargement resting on an apparently small pivot below and falling backwards or to the side from sheer attempt to get into a position of equilibrium—make an indelible impression on the observer. On examination of the head it is seen that the hair is slightly, if at all, developed, because of the loss of nutrition to the scalp, the epicranial veins are distended and shine through, the fontanelles are bulging and the seat sometimes of a bruit and always of a lifting impulse, and the whole cephalic extremity if placed between the eye and a strong light is more or less translucent.

If the hydrocephalus is present at birth and if it continues to develop with considerable rapidity there may be absolutely no awakening of the mental faculties. If it does not show itself until some months after birth (the rule), mental development will have begun to reveal itself; but the advent of the hydrocephalus stunts or holds it in check by the increase of fluid which usurps the place of the brain tissue. Not alone will the sequences of mental development be lacking, but all the psychophysical accomplishments, such as talking, walking, etc., will be delayed or perhaps prevented. On the other hand, if the hydrocephalus proceeds slowly, such children may be backward in learning to talk, to walk, and in getting their teeth, but eventually these take place while the higher mental faculties, such as association and apperception, are held in abeyance. That is, such children may develop intelligence and be in a mild way amenable to discipline, and may possibly be sent to school, but

generally they must be classified as idiots. Very rarely do we find one intellectual factor developed while the others remain dormant, as is sometimes seen in other forms of idiocy. The special senses are likewise very much undeveloped, and the sense of sight is generally lost on account of atrophy of the optic nerve incident to pressure, although in some cases sight may remain normal during the entire life of the child. The special sense that is least often affected is that of hearing.

When the hydrocephalus is well marked from the time of the child's birth, and of a progressive nature, motorial irritation symptoms will be very insignificant. When, however, it manifests itself some time after birth, these symptoms, such as spasms or convulsions constituting eclamptic attacks, spasticity, or complete paralysis of one half of the body may be present. The spasticity that occurs in chronic hydrocephalus affects the upper as well as the lower extremities and sometimes even the muscles of the body. Such symptoms are much more frequent with symptomatic hydrocephalus dating from or occurring soon after birth and dependent upon porencephaly and inflammatory lesions of the cortex, conditions which need to be carefully differentiated from genuine congenital hydrocephalus. In congenital spastic rigidity (Little's disease) the lower extremities alone are affected in any considerable degree. One of the most common irritation symptoms occurring in hydrocephalus is deviation of the axes of the eyeball, causing some form of strabismus, more rarely nystagmus. In acquired internal hydrocephalus, and in congenital hydrocephalus of delayed advent, there may be other inconstant symptoms, such as pain in various parts of the head, dizziness, vomiting, stiffness of the neck, intermittency of the symptoms, and other symptoms which sometimes prompt one to look upon them as of functional origin. Even in these cases the diagnosis is not materially furthered by these symptoms. The enlargement of the skull and the mental agenesis or deterioration are always the pathognomonic accompaniments.

Course and Prognosis.

The course of the disease, particularly in those cases in which the symptoms follow soon after birth, is generally uniformly progressive, and death follows simple exhaustion and asthenia in the first months of life, the infant really having had no life history; it has merely existed and passed away. In other cases, and particularly in those that reveal the hydrocephalus in the first and second years of infancy, the course of the disease is not uniformly progressive; it may be attended by periods of cessation from any active manifestation, even

increase of size, while at other times there is an elevation of temperature, vomiting, and increase in the frequency and disturbance in rhythm of the pulse, followed by prolonged stupor and rapid increase in the size of the head. These cases, in which the progress of the disease is inconstant, often succumb to the mildest attack of some intercurrent disease such as measles, bronchitis, or pertussis, while others are unable to resist such convulsive disorders as laryngismus stridulus, a condition which has been considered by some (von Ranke) a symptom of the disease, just as attacks of eclampsia—in which, by the way, these children very rarely die—are considered by many a symptomatic part of the disease. Very rarely does escape of the fluid through the parietes of the head or from the nares mark the progress of the disease, yet such cases have been put on record by Rokitansky, Nothnagel, Groh, and others. In these cases in which the escape of cerebrospinal fluid takes place through the nose it is probable that the avenue of escape is the perineural space of the olfactory nerves, the latter being atrophied, or it is barely possible that the ethmoid bone may become divorced from its attachments on account of the increased pressure which it is called upon to resist. The prognosis is fortunately very bad except in those cases in which the quantity of fluid is comparatively small, and the conditions on which it exists cease to be operative; in these the prognosis is excellent. There can be no doubt that a minor degree of hydrocephalus is no uncommon element in the life history of many children, and although it is surmised that most of these cases are of the acquired form, some of them are of the congenital. If the hydrocephalus is not so great as to subject the brain tissue to any great amount of pressure, or if it disappears before association and projection pathways take up their function, its occurrence in a mild form does not materially handicap the possessor. These cases are never recognized. When the disease corresponds with the description we have given, the sufferer is always a burden to his race. The prognosis as influenced by the treatment can be dismissed in a line. As yet the latter has not modified the former.

Morbid Anatomy.

When the skull cap is removed in a case of hydrocephalus the picture that is seen is a somewhat variable one. Ordinarily the meningeal coverings are stretched to their utmost; the cortex is robbed of its complex and distinguishing features; the convolutions and fissures appear like a distended bladder, on the surface of which are traced indistinct markings. No greater surprise can be given one than to

see a well-marked hydrocephalic encephalon. The limiting substance, remains of cortex and medullary tissue, may be reduced to a mere film, demonstrating that the brain proper (the highest level of Hughlings Jackson) has nothing to do with the maintenance of life. Of course the process may not be so far advanced, and then it still may be possible to distinguish the primary sulci and the rudiments of convolutions. But there is no differentiation even in these cases between white and gray matter. Occasionally one hemisphere will be in a much more advanced stage than the other, and cases have been reported in which the hydrocephalus was confined to one hemisphere, and, strange enough, the hemisphere on the other side was atrophied or rudimentary. It should not be forgotten that some cases of hydrocephalus present very slight change in the brain substance, simply distention of the ventricles and corresponding thinness of the hemisphere substance. In these cases the morbid accumulation of liquid takes place after the brain is well developed. When the ventricles are opened the accumulated fluid escapes in quantities varying from a few ounces up to fifteen quarts. An examination of this fluid shows that it is very similar to cerebrospinal fluid, that it contains the same element, and that it is more limpid. It has a specific gravity of a trifle more than water and contains a very small quantity of sodium salts and scarcely a trace of albumin. In exceptional cases the fluid has a slight color and contains a greater quantity of albumin. In these cases it is probable that the hydrocephalus has not been an uncomplicated one. When the interior of the brain is laid open it is seen that the ventricles are greatly distended, and in fact have lost all evidences of their normal confines. The vascular plexuses and tela choroidea are delicate and drawn out. The basal ganglia are flattened and robbed of their markings. The crura, the optic tracts, the pons, and all the structures at the base have lost their normal contour and relationship to a greater or lesser degree. The third ventricle is distended, in a less degree, of course, than the lateral ventricles, and frequently the aqueduct and fourth ventricle extending into the central canal are widely dilated. The cerebellum reveals changes corresponding to the amount of hydrocephalus. If the latter be very slight there may be only a lengthening of the tonsil and middle portion of the inferior lobe which projects like a spigot into the spinal canal with the oblongata, or the cerebellum may be pushed downwards towards the central canal following the displacement of the pons and the oblongata; the oblongata itself is often to be found reaching down to the middle of the body of the third cervical vertebra, or very rarely there may be such an intense degree of displacement of the cerebellum that it is pushed through the foramen magnum.

In one such case the distended and displaced cerebellum was mistaken for spina bifida cervicalis and operated on by Chiari. The cerebellum itself may be normal in texture, softened, or sclerosed.

When the ependyma of the ventricle and the choroid plexuses are examined carefully there will be found a certain amount of thickening and granulation which are considered to be of inflammatory origin. In conditions where the landmarks are so effectually obliterated as in this, delicate structures like the septum lucidum and the posterior commissure are entirely obliterated. The passageways leading from the lateral to the third ventricle, the foramen of Monro, and from the third to the fourth ventricle, ordinarily delicate and difficult to find, have been converted into widely dilated channels. Occasionally in unilateral hydrocephalus the foramen of Monro is found closed.

Examination of the cortex microscopically by means of the recent histological methods has been done in few instances. In these cases, however, the ganglion cells and their prolongations have been found small, atrophied, granular, pigmented, and stunted.

Treatment.

Hydrocephalus of rapid progress is entirely unamenable to every therapeutic procedure. In those instances in which it is of slight degree and advances slowly, partial or complete recovery sometimes follows. The desideratum in the treatment of all cases is to get rid of the fluid and prevent its production. For the former we have no hesitancy in recommending the procedure of puncturing the subarachnoid space in the lumbar region as advised by Quincke, a description of which is to be found in the article on meningitis. This is the only operative procedure that has ever been of the slightest service in my hands, and although yet unwilling because of limited experience, though such experience is favorable, to advise this measure as by far the most important, I feel inclined to say that of all surgical measures it is the one worthy of most confidence. Such measures as cranial puncture, tapping the ventricles, or trepano-puncture, and draining them, the introduction of saline solution or a water solution in which iodine has been mixed, and, in short, every mechanical procedure even to strapping and compressing the head—measures that are mentioned and often lukewarmly recommended by many authors—can be only unequivocally condemned. For a full discussion of the applicability of these various procedures the reader may be referred to the works of Broca and Maubrac and of Keen. My own experience teaches me to put in the same therapeutic category all drastic measures such as cathartics, diuretics, and

diaphoretics, administered with the mistaken idea that they contribute to the removal of the fluid; for, as rational interpretation of physiological teachings demonstrates that their tendency is to do the opposite, they are to be condemned. The little aid that lumbar puncture has to offer in these cases is greater in those that are postnatal in their development. Its efficaciousness in every case of congenital hydrocephalus is slight, and not at all to be compared with what is hoped will be its usefulness in symptomatic hydrocephalus. The treatment of this disease, aside from what may be called symptomatic, such as the administration of small doses of mercury in children whose parents have suffered previous syphilitic affection, or small doses of iodide of potassium in those in whom there is a marked antecedent diathetic condition, consists in the adoption of measures that contribute to a bettering of the child's nutrition. It is unnecessary to enumerate them in this connection. Children who have had a moderate degree of hydrocephalus and in whom the condition has come to a standstill, have often been seen to make encouraging physical and mental development when taken from the parents, placed in new environment, and subjected to wholesome discipline and an intelligent dietary. The education of these children should be carried out along those lines which are now adopted for the instruction of defective children by the advanced school of pedologists, and instituted principally by the elder Seguin.

A case that has been reported by Sourma is worthy of mention. He describes a case of congenital hydrocephalus, in which, through neglect, the child's head was frequently exposed to the direct rays of the sun. The author attributes the recovery to increased action of the sweat glands, a lame and unnecessary apology for a counterirritant.

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PARASITES OF THE BRAIN.

The occurrence of parasites of the brain is a very infrequent one, and especially is it so in this country. In Australia, however, and in Central and Southern Europe it is a condition which is sometimes seen during life and on the autopsy table. If a medical literature of Iceland and other polar regions existed, it is not at all unlikely that mention of its occurrence therein would be common. According to Thomas, of Adelaide, five per cent. out of a total of one thousand nine hundred and eighty-one cases of hydatid cysts were connected with the central nervous system. Only a brief consideration of the subject will be attempted here.

The principal parasites which invade the brain are the cysticercus cellulosæ and the echinococcus. Of these the first named is by far the most frequent and important. There must be mentioned another, which, although not properly classified as a parasite, but as one of the polymorphic bacteria, has been considered of parasitic nature, namely, the ray fungus or actinomyces. Böllinger, to whom next to Hahn we owe most of our knowledge of actinomyces, has observed one case of primary actinomycosis of the brain out of eighty-seven cases, the entire number studied.

The most important factors in the etiology of the parasitic diseases of the brain are uncleanliness, close contact with animals, such as the dog, and the ingestion of uncooked meat. The tænia echinococcus itself occurs exclusively in the intestine of the dog. Only the cyst worm occurs in men. This results from the entrance of tænia eggs into the intestinal canal, their maturation and the subsequent wandering of the embryo from the intestinal canal into some organ in which it changes into a cyst which is incapable of active motion. It follows naturally that in countries where the dog is the most important domestic animal, such as Iceland, or in countries where he is made use of as a beast of burden, such as Southern Germany, in-

fection with this parasite will be most common. The cyst which this parasite produces varies in size from a walnut to an apple. Endogenous or exogenous daughter cysts may develop and thus add very materially to the size. Very rarely the condition known as echinococcus multilocularis develops. In fact, Roth claims that the case which he describes is the only one on record. These are always very small cysts varying in size from a millet seed to a pea, but they are invariably present in large numbers.

Not infrequently the cysts, developing slowly and in more or less indifferent portions of the brain, do not produce any apparent symptoms for a long time, and in fact some cases are first recognized after death consequent upon some other disease. In other cases in which they develop more rapidly and in individualized parts of the brain, there will be not only focal irritation symptoms and pressure symptoms, but symptoms which are directly attributable to a local inflammation which exists around the cysts and which may be pathologically expressed by the formation of a connective-tissue capsule. In some very rare instances the cyst may cause sufficient pressure on the meninges to produce well-marked meningeal irritation symptoms, while in others, rarer yet, the same pressure may cause absorption of the bone sufficient to allow the little sacs to be seen peeping through, or, if on the base of the brain, to allow them to rupture into a cavity like the nasal cavity. Westphal has reported an example of the latter condition in which aspiration of a cyst revealed its true character, and after sixty such echinococci sacs had been emptied, cure of the disease, or at least its permanent amelioration, followed.

The cyst which is provided with a tapeworm head is known as a "measle" or cysticercus cellulosæ. These result from the boring of embryos through the stomach, and then by means of the circulation, and it is said also by migration, they reach their destination in the brain. There they undergo after a variable time changes into cysts, from the walls of which a scolex develops into the interior. These scolices have, when completely developed, a circle of hooks, suckers, etc., which are readily made out when seen under the microscope. The cysticercus cellulosæ, like the tænia solium from which it takes origin, is very rare in this country, more often found in Great Britain, and not at all uncommon in Prussia and Saxony. They may develop in the membranes of the brain, probably their most common location, and in the brain itself, particularly in the white matter. They may appear individually or in collections resembling a mulberry. In the latter instance they are known as cysticercus racemosus. These cysts are mostly sterile and like the cysts previously mentioned their importance from a physician's standpoint depends upon their location;

and what has been said about the previous cysts also applies here. Dressel has called attention to the very much more common infection of men than women by this parasite. The occurrence of actinomyces in the brain is such an extremely rare condition that it can be only referred to.

Although echinococcus forms in the brain a much larger tumor than cysticercus cellulosæ, each of these forms of cysts on reaching a certain size may undergo a retrograde change; that is, the liquid of the cysts becomes absorbed, the cyst wall shrivels, and all that remains is some fatty and caseous detritus, more rarely a slight amount of calcareous material, and perhaps some hooks. As in all other neoplastic conditions the cerebrum is more frequently the seat of the new growth than the cerebellum. In the cases collected by Thomas, the proportion of cerebrum to cerebellum was as fourteen to one. Of one hundred and fifty-six cases collected by Rosenthal, eighteen only were in the cerebellum. Though the proportion of cerebellum involvement has been found greater by some observers, such as Morgan, these figures may be taken as representing the general average.

Symptoms.

It is manifestly very difficult to depict any characteristic symptomatology of this disease. In some cases there will be no symptoms at all, in others there will be those most frequently found with the psychoses, such as hysteria, hypochondria, and acute mania, while in others still the symptoms will be very similar to those of brain tumor. Sometimes the symptoms will point to one of these conditions, not long after to another. As in other forms of new growth within the cranial cavity, the symptoms will depend upon the location of the cyst, upon the rapidity of its growth and its size, and upon whether or not it is enclosed in a sac of connective tissue, the result of its irritating presence. I have already referred to the fact that the cestode diseases have a special tendency to elect the cerebrum rather than the cerebellum, the proportion being about fourteen to one. It is well also to bear in mind, although no differentiation can be made in the symptomatology, that the echinococcus forms a much larger tumor in the brain than does cysticercus cellulosæ, and that the latter condition is by far the more common.

Frequently in the beginning the symptoms are very vacillating, unsuggestive, and indefinite. The most common early symptom is headache which, unlike the headache of brain tumor, is very apt to be intermittent. Although it may be of great severity, it is frequently of a dull aching character. With this, preceding or following it,

there may be evidences of psychical and motorial irritation, the former manifest by irritability, petulancy, loss of proper inhibition, or by apathy, stupidity, sleeplessness, and manifestations of mental enfeeblement, and the latter by local muscular twitchings or a generalized convulsive attack which may be of a simple epileptiform character or it may be more or less typically Jacksonian. These epileptiform fits may be of frequent occurrence and of considerable severity, and within or following them there often develops motor impairment which may reach complete hemiplegia with or without aphasia, paraplegia, or partial paralysis of one of the extremities, such as in a case reported by Mudd, in which there was partial paralysis of the forearm and hand combined with severe headache and choked discs. Paretic symptoms are by no means so common as convulsions. On the other hand there may be attacks in which only simple loss of consciousness can be made out, and these attacks may alternate or be followed by attacks of delirium which are so severe, and occasionally so continuous that the patient must be confined in an asylum. In some cases there are very strong focal symptoms in addition to those manifest during an attack of Jacksonian epilepsy—for instance, symptoms pointing to involvement of some of the eye muscles or impairment of one of the special senses, such as hearing, or if the cysticercus be in the fourth ventricle, disturbances of respiration, circulation, and of the glycogenetic functions, and even deep coma (Jaccoud).

If the seat of the cyst be the cerebellum, the symptoms will be of the same nature, but the headache will be very apt to be occipital or cervical, and disturbance of gait as well as manifestations of optic-nerve involvement, dizziness, vomiting, etc., will be of more constant and early occurrence. As a matter of fact, the disease, when located in the brain, if the patient is not a child, is not apt to produce symptoms of vomiting or optic neuritis early; nor, unless the meninges be involved, are gastric symptoms, elevation of temperature, post-cervical rigidity, and the like liable to form a part of the clinical picture. Involvement of the cranial nerves is not common, and when it does occur it is usually incomplete. A great number of cases is on record in which there was slight ptosis, some strabismus, involvement of the facial, etc. But these symptoms vary with each case and of course depend upon the locality, size, and number of the new growths.

The course of brain hydatids is not, like that of brain tumor, a uniformly progressive one, nor on the other hand is the patient liable to show entire freedom from all symptoms for a more or less prolonged time, as in hysteria, neurasthenia, etc. The severity of the symptoms may abate, but the patient will never be free from them. Headache and sleeplessness and irritability will be followed by delirium, apathy,

or dementia. Focal epileptiform attacks will be followed by motor paralysis, and the growth of the cyst in the brain will produce the well-known concomitants of intracranial pressure. Choked discs are frequently to be made out by the ophthalmoscope. In a few cases that have been reported, symptoms referable to the spinal cord have been found. Clarke has published a case in which there was hemiplegia and wasting of the paralyzed limb. Microscopical examination showed changes of disorganization in the ganglionic cells of the corresponding cornua of the spinal cord. Oppenheim has reported a case of cysticercus of the fourth ventricle in which there was paralysis of all four extremities but which recovered so far as to leave only a paralysis of the legs and other clinical manifestations indicative of a lesion in the spinal cord, but no lesion was discovered. In cases in which spinal-cord symptoms are well marked or predominate, it is quite probable that there may be coincident infection of the brain and cord.

Diagnosis.

There is nothing characteristic in the symptomatology, and we must look to other factors for aid in diagnosis. The trouble can scarcely be suspected until evidence of the presence of the parasites is found in other parts of the body. The most common situation is in the skin. Here there may sometimes be seen and felt small circumscribed elevations, just beneath the skin or deeper, of the size of a pea, sometimes smaller, very rarely larger, and of an indescribable elastic resistance, not sufficiently great to suggest a small fat tumor, nor yet sufficiently small to give the impression of a cyst. If these are found coincident with such symptoms of brain involvement as we have mentioned, and an examination of their contents reveals the presence of scolices or any other integral part of these parasites, the diagnosis is made. On the other hand, if an ophthalmoscopic examination reveal them in the choroid, as it sometimes will, the diagnosis is unassailably made. If the cyst in the brain produces such absorption or elevation of bone by pressure that a needle can be passed into one of the cysts, or the elevated bone removed, their contents evacuated and examined, the diagnosis might be reached in this way as it was in the case reported by Mudd; or if the contents of the cyst get into the system of ventricles, which they do very rarely, examination of a fluid obtained by puncture of the lumbar region in the subarachnoid space may be the means of prompting the diagnosis. Where all these aids to diagnosis are lacking, one can scarcely suspect the real nature of the disease.

In some cases the diagnosis is not only not made but the disease

is not even suspected. This is illustrated by a case reported by Simmonds in which a large number of cysts, containing the hooklets of cysticercus cellulosæ, were found after death in the brain of a patient who was supposed to have suffered only from simple dementia and general marasmus.

Differential Diagnosis.

I have said that the diagnosis will be facilitated if it be borne in mind that the chief symptoms, in the beginning at least, are irritative headache and cortical epilepsy, and that the disease runs an afebrile course. Yet these are not uncommon attributes of other brain tumors, and it is only possible to suspect cysticercus after long-continued observation. Malignant growths in the brain are characterized by a more uniform course, while gliomata are usually attended by more severe symptoms than cysticercus. Cysticerci of the brain progress slowly, and the course of the disease is attended by exacerbations of convulsions, headache, and dizziness, which may be followed by more or less prolonged periods of comparative comfort. Isolated tubercles of the brain, with which cysticerci are most often confounded on account of the protracted course of each, are attended more constantly with pain, headache, and dizziness.

Prognosis.

The prognosis of parasitic affections of the brain is always very unfavorable. That they do not, however, lead so unerringly to death as do brain tumors, may be gathered from the remarks which we have already made regarding the change that may take place in the contents of a cyst and the changes which the cyst may cause in the walls of the skull. The number of cases, however, which go on to spontaneous recovery or which cause changes that may lead to recovery is very small compared with the actual number of existing cases. It is impossible to say just how long the disease may exist, but from two to four years is the average duration. On the other hand the prospects which treatment can offer are not material, so, taking it all together, the prognosis is as unfavorable as it can easily be; fully as bad as in any other form of brain neoplasm. The disease makes interrupted progress, and death is usually by convulsions.

Treatment.

This is one of the diseases which prophylactic treatment should completely stamp out. The prevention of dogs and human beings

from being housed under the same roof, the disposal of the offal of dogs so that it does not contaminate the water or food supply of man, the interdiction of uncooked meat as an article of diet, and the enforcement of the hygienic and sanitary conditions of modern civilization will effectually eradicate the disease.

When the disease does occur and when it can be diagnosed and localized, it should be subjected to the same treatment as any other form of intracranial growth. Unfortunately, on account of the multiplicity of the cysts and their locations in different parts of the brain, surgical measures do not hold out the same hope of relief that they do in superficial and localizable neoplasms of a different nature, even though that be small. On the other hand it may be said that if the nature of the trouble be suspected, early operation is more justifiable in this disease than in any other neoplastic condition. The great number of cases, however, will still continue to go to the insane asylums.

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THROMBOSIS OF THE DURAL SINUSES.

The intracranial sinuses are wide, rigid tubes between the two walls of the dura which receive the return flow of blood from the brain and pour it into the jugular veins. They are essentially venous in their structure, though devoid of valves, and are liable to the same diseases as the veins. They differ from the veins in action, insomuch as their calibre always remains the same, and particularly that it is not subject to any variation during respiration.

The pathological conditions that occur in the sinuses are those that are seen in the veins, namely, simple coagulation of the blood, not necessarily accompanied by any change in the walls of the sinus, and inflammation of the walls of the sinus with resulting coagulation, constituting a genuine phlebitis. The former occurs with conditions of profound hæmolysis, the latter with local adjacent and general pyogenic infection. These two conditions differ in their genesis, and an attempt is made to discriminate between them clinically.

Thrombosis of the dural sinuses was one of the first intracranial diseases to be recognized and differentiated. English surgeons particularly mention many examples of it, and Abercrombie has recorded an example which he observed in 1816. Ophthalmologists have materially contributed to the elucidation of the etiology and pathology of thrombosis of the cavernous sinuses, particularly Knapp, of New York, who was the first to recognize the relationship of disease of the eye to inflammation of the ophthalmic veins. The subject has lately been given thorough consideration from a retrospective and

compilatory standpoint by Lancial, and from the standpoint of an extensive experience, clinical and anatomical, by Macewen.

Etiology.

The causes of thrombosis of the dural sinuses may be considered under three heads: First, causes operative through the blood alone; second, causes operative through infection and inflammation of the sinus walls; and third, pressure on the sinus, as by a tumor. Thrombosis which occurs as the result of blood depravity is known as marasmic or autochthonic. It is a very uncommon disease, and rarely occurs except in new-born and in young infants predisposed to it by choleraic disorders and the acute infectious diseases, by inherited syphilis, by rachitis, by pulmonary tuberculosis, or in short by any diseased condition that causes profound and rapid depreciation of nutrition. It sometimes occurs at the other extreme of life, in those immediately predisposed to it by causes not unlike those operative in infancy, as well as by diseases common at that time of life, such as cancer. Very rarely it develops during the puerperal state. Böllinger has shown that it occurs sometimes in chlorotic girls and in adults without discoverable cause, as in cases reported by Bucklers. Impaired activity of the heart is always an active predisposing cause. In some instances primary anæmia after profound loss of blood is the only discoverable cause, and v. Dusch has reported two cases due to this.

The infective or inflammatory forms occur at a different period of life from the marasmic form—almost entirely during youth and adult life. Infection of the sinuses is the result of contiguous septic inflammation from the dura or from the bone which has become diseased as the result of injury and infection, or infection alone. The longitudinal sinus is most frequently the seat of disease from such causes. The most frequent cause of pyogenic sinus thrombosis is a purulent affection of the middle ear and coexisting caries of the petrous portion of the temporal bone, the sinus most likely to infection in the latter being the lateral and especially that part of it contained in the sigmoid groove, sometimes called on that account the sigmoid sinus. The petrosal sinuses are less often affected. Extension of inflammation from the ophthalmic veins, the original source being from the eyes and the orbits, is the most common cause of thrombosis in the cavernous sinus. Any pyogenic inflammation of the head and neck in the area drained by the cerebral veins may be the starting-point of the process that eventually causes thrombosis of a dural sinus. It is found associated with erysipelas, with glanders, with angina Ludo-

vici, with suppurative tonsillitis and retropharyngeal abscess, pyogenic and destructive processes in the nasal cavity, with alveolar abscesses, and with epidemic parotiditis. Sinus thrombosis arising from these latter factors is frequently preceded and accompanied by phlebitis of the temporal and facial veins, but not necessarily so. The sinus commonly involved from these extracranial causes is the cavernous. A case has been published by Pilcher in which septic sinus thrombosis followed symptoms of chills, vomiting, and diarrhœa, the immediate consequence of eating gluttonously of decayed goat's meat.

Sinus thrombosis may result from pressure on the walls of the sinus which causes constant impediment to the circulation of the blood. Such conditions are occasionally furnished by brain tumors (as in a case reported by Bucklers), brain abscess, depressed fracture, and pachymeningitis. The superior longitudinal sinus is most liable to this form of involvement. Thrombosis that results from brain abscess is, however, almost always dependent upon the same causes as the abscess itself.

Pathology.

The immediate preceding cause of marasmic sinus thrombosis is a relative dissociation of the constituents of the blood—a great depreciation of nutrition and relative increase of the fibrin and its antecedents, the white corpuscles, and especially the blood plates. This, combined with the loss of circulatory vigor, increases the sluggishness of the return circulation, never lively in the longitudinal sinus, and taken in connection with the anatomical peculiarities of the sinus, its angularity and its trabeculæ, directly contributes to the immediate occurrence of coagulation. In those cases in which it follows exhaustive diarrhœa, as it does so often in young children, not alone the serum of the blood becomes enormously diminished, but the serous fluids of the body as well, a state of profound oligocythæmia. The lymph circulation is in a state of stasis, and this is soon followed by thrombosis in the longitudinal sinus. The thrombus itself is seen to have the characteristics of other marasmic thrombi. It is dense, does not crumble easily, is somewhat irregular in extent, and in some cases, if the factors leading up to the disease have been profound, thrombi will be found in all the sinuses and in the jugular veins. If they have been very slow in forming, they have a stratified appearance, showing that the various layers have coagulated at different times, and they may have become organized. In some cases marasmic thrombosis is of such intensity that the meningeal vessels become so distended that hemorrhage occurs. Such hemorrhages

occurring during the prenatal period or in the new-born cause one form of porencephaly.

The pathological changes in sinuses, the seat of inflammatory thrombosis, are similar to those of infective inflammation in other vascular structures. The endothelium of the inner coating is percolated by the infectious element and the exudate produced by it; it soon detaches itself and the walls of the sinus become more deeply involved. The process in the sinus walls may go on to extensive ulceration and destruction. Simultaneously with the desquamation of the internal coat and the exudation into the walls, the current of blood through the sinus becomes slowed, the trabeculæ which stretch across the sinus act as foreign bodies, and coagulation of the sinus' contents occurs. The clot thus formed then becomes infected if the source of the original infection is very active, and at the same time the exudation from the walls of the sinus extending into the surrounding tissue, the bone on the outside and the meninges on the inside, forms a distinct suppurative mass. The coagulated contents of the sinus, if they could be seen at this time, would vary in appearance according to the intensity of the infection and the variety of cocci that are present. Usually, however, they consist of a dark, pasty, reddish mass, having a tendency to disintegration. If bits of this disintegrating infectious mass get carried into the circulation, metastatic septic phenomena soon occur. The obstruction of the sinus is complete. This causes a very marked hyperæmia in the veins which are confluents of the sinus. If the inflammatory process be very severe, it may call forth meningeal hemorrhage, but this is not very common, nor is cerebral softening, unless there be secondary pyogenic infection of a localized brain area, when brain abscess develops. The damming back of the return circulation of the brain often causes a considerable degree of hydrocephalus. The venous territory that may become infected from a severe pyogenic focus in a sinus is often very great; not alone the sinuses which empty into the one infected may contain thrombi, but the neighboring veins as well. Thus, the internal jugular is sometimes found to contain a coagulum. The septic organisms may gain entrance into the blood, and when they do metastatic abscesses in remote organs, such as the kidney, the lungs, and liver, sometimes develop. When the purulent process extends to the meninges or the substance of the brain, the immediate results are purulent meningitis, or brain abscess. When the infective process is not of sufficient severity to cause destruction or disintegration of the walls of the sinus, the clot which forms may become organized by the development in it of vascular buddings and proliferations which arise from it and from the adjacent dura. This process of organiza-

tion may completely occlude the lumen of the vessel. But even from such a coagulum as this, small pieces may be detached and passed through the return circulation, acting as infection carriers to distant parts.

The salient points of difference between marasmic and infective thrombosis are summed up by Macewen as follows:

Marasmic.	Infective.
1. Chiefly affects the azygos sinuses.	1. Chiefly affects the dural sinuses.
2. The clots tend to organization or are absorbed.	2. Clots tend to purulent disintegration.
3. Hemorrhages into cerebral cortex in about half the cases.	3. Hemorrhages into the brain or cerebellum seldom occur.
4. Tendency to produce brain softening.	4. No tendency to brain softening.
5. There is seldom purulent infection as a sequence.	5. Purulent infection common; septic or infective emboli.
6. No accompanying leptomeningitis nor cerebral or cerebellar abscess.	6. Often coincident purulent leptomeningitis; cerebral or cerebellar abscess.

Symptoms.

The symptoms of sinus thrombosis vary with its causation and location. In the marasmic form they often come on very slowly and are so completely masked by the symptoms of the disease that calls it forth that they pass unrecognized and are discovered, if at all, on the post-mortem table. For instance, a case of marasmic thrombosis occurring in a young infant sequentially to a sharp attack of cholera morbus causes no symptoms which may not be considered a part of that disease, except, possibly, a profound sluggishness of cerebral functions, a tendency to collapse or to coma. The same may be said of this variety of thrombosis occurring in the senile, following on profoundly debilitating diseases. If it be borne in mind that marasmic thrombosis does not always occlude completely the calibre of the sinus, that it forms very slowly, and tends quickly to organization, this fact will be readily understood.

Infective thrombosis, on the other hand, comes on very abruptly and is attended with symptoms which indicate a profound septic infection. These are: chill, or recurring sensations of frigidity, abrupt rise of temperature, headache, nausea and vomiting, and a feeling of profound unrest.

Marasmic thrombosis may, however, be accompanied by symptoms pointing to obstruction in the longitudinal sinus, the favorite seat of this form. These symptoms are: swelling over the frontal sinuses, distention of the frontal and temporal veins, circumscribed

cyanosis of the face in the territory drained by the anterior facial veins, epistaxis, continuous or recurring, slight and variable amount of squint, sleeplessness or mental hebetude which may deepen into coma, or which may be interrupted by universal or unilateral convulsions, sometimes followed by contracture of an extremity, or paralysis. In young children the convulsions may be prominent symptoms, but in adults they do not usually play a very important rôle. If the disease occurs before the fontanelles are closed, there may be depression over them in the beginning and a bulging later, the latter due to a hydrocephaloid condition. In the adult, the headache and cerebral torpor, sleeplessness, delirium and coma, coming on towards the end of any exhausting disease, are symptoms of great gravity and usually indicate marasmic thrombosis. Occasionally there is considerable premortal rise of temperature, although in the beginning and during the course of the disease the temperature is subnormal. The febrile phenomena may be due to secondary diseases, pneumonia, decubitus, etc.

In the infective form the general symptoms in the beginning may be, as we have already mentioned, those indicative of profound septic infection. When such symptoms occur in the course of any septic disease, such as otitis media, erysipelas of the head and neck, phlegmon of the eye or orbit, or purulent disease of any of the cavities of the face, infective sinus thrombosis should be suspected. After the initiatory symptoms the clinical picture that develops does not differ from that of ordinary septicæmia, except by the addition of localizing brain symptoms. According to Macewen, the symptoms may assume a pulmonary form, a gastrointestinal form, and a cerebral form. In the pulmonary form, in addition to the temperature, rapid, feeble pulse, profuse sweats, slightly jaundiced skin, scant, high-colored urine, and sluggishness of the alimentary tract, symptoms of involvement of the lung, such as cough, profuse fetid expectoration, and dyspnœa, are so prominent that attention is directed to the lungs, and frequently at the expense of the original seat of the disease. In the gastrointestinal form, the symptoms are those of diarrhœa, foul breath and tongue, and a rapidly developing typhoid state, in addition to the general symptoms mentioned of the preceding form. Accompanied or not by these pulmonary and gastrointestinal symptoms pointing to general septic involvement, headache, excitability and irascibility, delirium, somnolency, and coma develop, and are often attended with such irritation symptoms as localized convulsions, strabismus, contracture of the extremities, and irregularity of the pulse. Other symptoms that cause variations in the clinical picture depend largely upon the sinus which is obstructed. Those associated

with thrombosis of the superior longitudinal sinus have already been mentioned under the symptomatology of marasmic thrombosis. They do not differ when this sinus is the seat of infective thrombosis, except that their intensity is greater.

Thrombosis of the cavernous sinus produces such local symptoms as bring these cases often under the care of the oculist. These symptoms are exophthalmus, corresponding to the side which is the seat of the thrombus, and caused immediately by stasis of the retrobulbar veins. This is accompanied by chemosis of the lid, proptosis and œdema of the orbital tissues, all determined by the stasis in the ophthalmic veins. The pupils are contracted, and if intraocular examination is made, there will often be found a stasis in the venous capillaries of the retina and a condition of the discs resembling papillitis. This condition is naturally accompanied by obscuration of vision. With this form there are sometimes seen circumscribed areas of the forehead and face which are covered with sweat and which have been thought to be quite diagnostic. In a few cases paralytic third-nerve manifestations, such as a mild degree of ptosis, are present, but on account of the intense œdema are scarcely to be made out. But when the involvement of other fibres of the third nerve is present, the paralysis of the ocular muscles will show itself by deviation of the ocular axis. Very rarely the fourth and the sensory branches of the fifth are implicated. When they are involved, symptoms indicative of their disturbed functions will be added. In the beginning the ocular symptoms are unilateral. As the disease progresses and the area of thrombosis extends, the other eye may become involved, and that while the severer manifestations are subsiding in the eye where they first showed themselves. Towards the end of this form of the disease, symptoms pointing to involvement of the basilar meninges rarely fail to show themselves.

Thrombosis of the sigmoid sinus is seen more often by the aurist, as the common cause of thrombosis in this location is infection from the middle ear. Often the first symptoms are cessation of discharge from the ear, accompanied by pain in the ear and head of increasing severity, and the usual symptoms of cerebral irritation. These are soon followed by three local symptoms which are looked upon as of great diagnostic importance. These are swelling of the mastoid region, enlargement of the cervical glands, and pain on pressure and percussion behind the ear. These symptoms are all produced by distention and obstruction of the sinus and of the mastoid and cervical veins, superficial and deep. When the jugular vein becomes thrombosed, it assumes the same whipcord-like rigidity as phlebitis of other veins and is accompanied by pain and œdema. After symptoms of throm-

bosis in the transverse sinus have been present for some time, there rarely fail to develop the clinical phenomena attending basilar meningitis, and these symptoms are often the immediate precursors of death.

Course, Duration, and Prognosis.

The course of the marasmic variety may be intermittent. It depends upon the severity of its causative factors. The clot may become organized and the patient may make, at least, a partial recovery. Very rarely does he recover so fully that no appreciable mental or physical defect remains, although it should be stated that Wernicke in his text-book states that primary thromboses of individual sinuses are of favorable prognosis. In one instance the sequela may be limitation of mental development; in another choreic movements, bilateral athetosis, infantile cerebral palsy, or a localized cortical epilepsy. A case of thrombosis of the longitudinal sinus and of the anterior frontal vein, causing circumscribed foci of hemorrhage which produced remarkably localized cortical epilepsy, has been recorded by Horsley. As a rule the marasmic form, if uninfluenced by treatment, leads to a fatal termination, especially when the etiological factors are of great severity.

The course of the inflammatory or infective form of thrombosis is progressive unless limited by early operative procedure. Death is the common termination, although resorption of the clot does take place in a few instances when the infection is a mild one. The duration of the disease varies from a week to a month, very rarely longer. Symptoms indicating involvement of distant organs by infected emboli having their origin in the clot lend the greatest gravity to the prognosis. When occlusion of the sinus by the clot is complete, septic infection of the general system is slow, and not so severe as when the occlusion is partial. In short, it may be stated that every case in which the disease is allowed to continue until disintegration of the clot occurs before the aid of the surgeon is implored, is beyond the help of therapy.

Diagnosis.

The diagnosis of sinus thrombosis is not difficult when the diseases which cause it do not obscure its individual symptoms and when the local symptoms are moderately well pronounced. If a patient develops such general brain symptoms as irritability, delirium, somnolency, convulsions, at the end of an acute infectious or exhausting disease, or if such symptoms are developed in a patient suffering

from a local purulent disease like otorrhœa, the presence of sinus thrombosis should be suspected. Intense headache and vomiting are not so significant of the disease as they are of meningitis and abscess. The rapid occurrence of such local symptoms as those already mentioned, pointing to involvement of the different sinuses, will then make the diagnosis of the disease a certainty. The two conditions with which it is liable to be developed are abscess of the brain and purulent meningitis, possibly also tubercular meningitis, for that may simulate almost any acute or subacute intracranial affection. The general symptoms will aid in excluding the three last mentioned.

Treatment.

The most important treatment and the one to which our best efforts should be directed, is preventive. Just so long as the rank and file of the profession take the same view of the innocuousness of slight and perhaps intermittent purulent discharge from the ear as do the laity, just so long will infective sinus thrombosis demand its quota of innocent victims. There is no greater need for reform in any department of the hygiene of childhood than in the treatment of diseases of the middle ear, which are so common in children of the ordinary walks of life, following the infectious diseases. When any of the etiological factors which we have enumerated for the infective form are present, the fact that they may eventually cause sinus thrombosis should be kept in mind and proper measures for prevention be taken. The modes and means which may be utilized to prevent the marasmic form are evident. They are directed entirely to overcoming the acute and profound nutritional changes which take place in the blood, whether they be anæmia, chlorosis, hæmolysis, or oligocythæmia. The treatment of the infective form may be summed up in one word—operative. The assistance of the surgeon should be invoked early, and he should be urged to operate. The status of cerebral surgery to-day points unequivocally to the fact that the most productive fields for the surgeon's efforts are in the pyogenic diseases of the sinuses, of the membranes, and of the brain. Compared with the usefulness of such aid in brain tumors and in epilepsy, the advantage is overwhelmingly with the former. The experience of one British surgeon alone, Macewen, gives tenor to this statement. The surgical technic of the operation need not be detailed here. Briefly it consists in emptying the middle ear, antrum, and mastoid cells of inflammatory products; exposure of the sinus; then opening and curetting, with or without previous ligation of the jugular vein. In some cases in which the diagnosis is

doubtful, the sinus may be explored with a sterilized hypodermic needle before chiselling into it. All writers seem to be agreed that the gouge is a safer instrument than the trephine. After the sinus is laid open and the inflected mass removed, the interior of the sinus should be made as far as possible aseptic, and then should be stuffed with iodoform gauze; precautions being taken to prevent the entrance of air into the sinus. The experience of Horsley, Ballance, Zaufal, Lane, and others has shown the advisability of previous ligation of the internal jugular vein before the lateral sinus is exposed, but that this is not always necessary is shown by a case recently reported by Adams, in which uninterrupted recovery followed laying open and curetting the lateral sinus.

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DISEASES OF THE CEREBELLUM.

Anatomy.

From the standpoint of anatomy and histology great progress has been made during the last few years in our understanding of the cerebellum, nevertheless the functions of it, the physiology in other

words, is still very obscure, and this despite the enormous amount of work that has lately been done to solve the problem and notwithstanding the publications of such contributions as those of Luciani, Ferrier, Marchi, Russell, *et al.*

In the brief consideration which can here be given of the subject I shall confine myself principally to the symptomatology of cerebellar disease.

The cerebellum, situated in the posterior fossa of the skull, comprises two lateral hemispheres and an inferior central portion called the worm or vermiform process. The latter which is sometimes spoken of as composed of a superior and inferior worm, connects one hemisphere with the other.

The obvious connection of the cerebellum with other portions of the encephalon is by three peduncles, the superior, middle, and inferior. The inferior peduncles are formed of upward continuation of the restiform bodies, the middle by the ventral, transversely coursing fibres of the pons. The upper portions of the superior peduncles pass from the medullary substance of the hemispheres and run upwards and forwards towards the corpora quadrigemina under which they pass. The superior cerebellar peduncle is connected with the red nucleus and pulvinar of the opposite side, but the decussation is not complete. The middle peduncles are not alone commissural fibres between the cerebellar hemispheres; some of their fibres pass into the nucleus reticularis tegmenti and the gray matter of the pons of the opposite side. The posterior longitudinal bundle and Reil's fascicle of the fillet leave the cerebellum by way of the middle cerebellar peduncle and give the cerebellum connection with the cranial nerves, the gray substance of the pons, the corpora quadrigemina, and possibly the corpora striata. The inferior cerebellar peduncles connect the cerebellum with the lateral columns of the oblongata and the nucleus of the same side, the inferior olivary body of the opposite side, and the nuclei of the posterior columns of both sides.

In architectural constitution the cerebellum resembles the brain, the gray matter being on the outside, the white within. The gray matter is involuted beneath the surface into numberless closely placed fissures which run transversely, nearly encircling each hemisphere. The fissures of the cerebellum do not extend very deeply beneath the surface, with the one exception of the horizontal fissure which divides the organ into an upper and lower portion.

The upper portion of the worm is made up of fine lobules which are called, enumerating from above and in front, the lingula, the central lobe, the culmen, the clivus, and the folium cacuminis. The upper portion of the hemisphere is also divided into four or five por-

tions corresponding to the divisions of the lingula, but these subdivisions are of less importance. They are known as the posterior superior lobe, the posterior and anterior crescentic, and the ala lobuli centralis. Every lobe is in continuous connection with the corresponding portion of the other hemisphere by means of a part of the worm.

The under surface of the worm presents four lobules—the nodule, the uvula, the pyramid, and tuber valvulæ. The connection of these with the constituents of the cerebellar hemisphere on the inferior

FIG. 17.—The Constituent Elements of the Cerebellar Cortex (Schematic).

surface is not so intimate as on the upper. The lobes of the under surface of the hemispheres are called the flocculus, the tonsil, the biventral lobe, the slender lobe, and the inferior semilunar.

When the cerebellum is cut through in the longitudinal axis, the cut surface has an appearance similar to a branch of a fir tree, the arbor vitæ cerebelli, with the stem extending upwards and anteriorly over the roof of the fourth ventricle. The gray matter is involuted about and indentated into the many peripheral ramifications of the white matter. The gray matter, however, is not entirely on the periphery, a number of circumscribed collections, nuclei, which are

named the nucleus dentatus, nucleus emboliformis, nucleus globosus, and nucleus of the root or nucleus castigii, being found embedded in the white matter. Of these it has been remarked that the nucleus lying in the inner part of the hemisphere bears a close resemblance to the olivary body of the oblongata.

The gray matter of the cerebellum consists of two layers, the outer or molecular layer, the inner or granule layer, between which is a layer of large nerve cells, the cells or corpuscles of Purkinje (Fig. 18). The cells of Purkinje are large flask-shaped cells, with a number of dendritic processes which ramify to a large extent in the outer or molecular layer of the gray matter, and an axis-cylinder process, or neuraxon, which passes into the granule layer, where it becomes medullated, and then continues with the medullary white matter beneath.

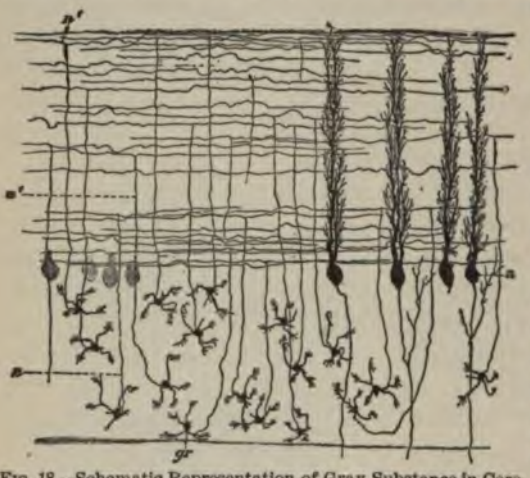

FIG. 18.—Schematic Representation of Gray Substance in Cerebellar Convolution. *gr.*, Granula; *n*, its nerve process; *n'*, divisions of the latter in the molecular layer, each separating into two fine longitudinal fibres; *p*, cells of Purkinje.

Into a more detailed description of the microscopical structure of this very interesting portion of the nervous system it is impossible to go. The reader who desires to become familiar with its intricacies, so far as yet made out, is referred to the recent writings of Kölliker, of S. R. Cajal, and to the admirable review of Schäfer in the third volume of Quain's "Anatomy."

Functions of the Cerebellum.

Since the time of Flourens and Magendie's classical experiments on the cerebellum, physiologists have been endeavoring to learn its functions by interpreting the results of partial and complete destruction and by extirpation. They, however, had gone but little further in solving the riddle than did their illustrious predecessors, until they invoked the aid of the pathologist and the clinician. The former has employed the methods of secondary degeneration, assisted by the technic of Marchi, to great advantage; while the latter has care-

fully noted the symptoms of disease of the cerebellum, such as apoplexy, sclerotic atrophy, and tumors, particularly in reference to the individual parts of the organ involved, and connated them with post-mortem findings.

Partial destruction of a cerebellar hemisphere is known to cause, according to the amount of tissue removed, want of harmony of muscular movement, irregular and violent agitation, complete loss of power to direct purposive movements, and asthenia amounting in some cases to a state analogous to paralysis. When one-half of the cerebellum is removed, degeneration occurs in all three peduncles of the same side, but none in the peduncles of the opposite side. Some degenerative fibres are seen passing along with the roots of nearly all of the cranial nerves. Extirpation of one-half of the worm produces some degeneration in all of the peduncles of that side. The degeneration in the superior peduncle is rather slight, and the degenerated fibres all cross to the nucleus tegmenti of the opposite side. The degeneration of the fibres of the middle peduncle is most marked in the upper third of the pons, and degeneration of the inferior peduncle resulting from such an extirpation is confined to the lateral part of the restiform bodies, and to those fibres of this peduncle which have their origin in the cerebellum and which go to the inferior olive of the opposite side. Others still pass into the fillet and to the posterior longitudinal bundle, and through it to some of the cranial nerve roots, particularly the third, the fifth, the eighth, and the twelfth.

It is of the greatest importance that these degenerations resulting from hemi-extirpation be borne in mind when we come to interpret the symptomatology of cerebellar disease, particularly the symptom which has been called by Luciani cerebellar asthenia, a term which I shall later make use of.

The theories that have been formulated concerning the functions of the cerebellum are ingenious and innumerable. It is neither wise nor necessary to cite them all here. Gall believed that it was the seat of the sexual instinct, Rienzi that it was the storehouse of the memories, and Foville, and before him Lusanna, taught that it was the organ of muscular sense. Folet has recently elaborated a theory which posits the cerebrum as the organ of intellectual life, the cerebellum the organ of the emotional part of psychical activity; the cerebrum and ventral spinal cord forming the motor order of the nervous system, the cerebellum and posterior spinal cord the sensory. Fanciful as such a theory may seem on casual consideration, it is quite astounding what a wealth of reasoning and contributory evidence it contains when examined into closely.

There can be no doubt that the cerebellum is the essential organ

for the coördination of voluntary movements, and that destruction of it and of its components produces ataxia of all such movements. It does not produce loss of muscle power in the strict sense of the term; that is, it does not produce paralysis, nor disturbances of sensibility, nor intellectual impairment. Its function is bilateral, predominantly equilateral, and its action extends to all voluntary muscles. The view of Luciani that the cerebellum is not an intermediate organ intercalated in the cerebrospinal system, but a terminal organ connected directly with the gray substance and centres of the cerebrospinal system, and incidentally with the peripheral motor end organs, seems to me a very plausible one.

Time has consecrated the opinion that the worm has a different or a more highly differentiated function than the lateral lobes of the cerebellum. This is denied by Luciani, who believes that every part of the organ is functionally homogeneous, and that loss of the worm can be "organically compensated" by the lateral lobes. He assumes that the functions of the cerebellum are a strength-increasing sthenic action (azione sthenica), tonic action (azione tonica), and static action (azione statica), *i.e.*, an increase of the impulses during the functional activity of the muscles and regulation of the normal extent and serial order of the movements.

Symptomatology of Cerebellar Diseases.

Disease of the cerebellum is attended by the development of certain symptoms in a certain way so constantly that cerebellar symptoms have come to be looked upon as forming a class by themselves. These symptoms are ataxia, incoördination of voluntary movements, staggering, reeling gait, headache, vomiting, choked discs, optic neuritis, vertigo, pain and tenderness in the region of the occiput, muscular weakness or asthenia, disorder of the deep reflexes, convulsions, tremor, nuclear and root palsies of the cranial nerves, polyuria, glycosuria, tachycardia, trophic disturbances, and mental impairment. Some of these symptoms are not directly the result of disease of the cerebellum; they indicate disturbance of intracranial vascularity and pressure which the cerebellar disease causes. To illustrate, Ackermann has published an example of gliosarcoma of the superior worm in which the chief symptom was spastic ataxia and paralysis of the extremities. To claim that this condition was a symptom of disease of the vermis, except indirectly on account of the enormous hydrops of the lateral ventricles which it produced, would be absurd.

The diseases affecting the cerebellum that we are as yet familiar

with, are tumors, abscess, sclerotic atrophy, and congenital and developmental defects, although the cerebellum may be the seat of morbid changes, similar to those occurring in other parts of the brain, in encephalitis, meningoencephalitis, and multiple sclerosis. The latter conditions affecting the cerebellum never occur alone, and their presence can only be suspected when there are superadded to the general symptoms of these diseases some "cerebellar" symptoms. Therefore, what is said here in reference to the symptomatology of cerebellar disease is based largely on the symptoms of the three first-named diseases.

Headache and vertigo are the most constant symptoms of cerebellar disease, but there is nothing in the location, in the intensity, or in the character of the headache to suggest that it is due to disease of the cerebellum and not to other parts of the brain. It may be of one side of the head, of the occipital region, or of the supraorbital region. When disease of the cerebellum which produces the headache is situated superficially and when it is of rapid evolution, pain in the occipital region is more common than in any other part of the head. Vomiting, choked discs, optic neuritis, and vertigo are, like headache, symptoms common to all forms of intracranial disease that disturb the circulation and rapidly increase intracranial pressure. Their occurrence with disease of the cerebellum is more constant than with involvement of any other portion of the encephalon, and the early appearance of choked discs and the intense degree which the optic neuritis reaches within a short time are to be considered very suggestive of cerebellar involvement. Taken in connection with other symptoms, such as ataxia, staggering, reeling gait, instability of the body in station and in motion, they are considered pathognomonic of cerebellar disease. The vertigo which is such a common symptom is so pronounced in upward of one-half of the cases to make it a principal complaint on the part of the patient. Like the headache, it has no special characteristics; it seems to be associated with the vomiting, or, better said, the vomiting seems to follow severe spells of vertigo, as in seasickness.

The disturbance of gait or of locomotion in cerebellar disease is the symptom of grand importance, and although absence of the characteristic gait has been noted in a number of cases of cerebellar disease, particularly of tumor, this in no way militates against the truth of the statement that cerebellar gait is by all means the most important diagnostic symptom. A proportion of those cases in which it has been reported absent are to be explained by the fact that the disease was of extremely slow progress, and that the symptoms of ataxia were probably present very early in the disease before the

patient came under the notice of the physician and before other parts of the cerebellum compensated for the parts that were being destroyed slowly. The ataxia of cerebellar disease varies according to which portion of the reflex arc, the motor or sensory, is diseased. In one case the defect of gait is similar to the uncertainty of one inebriated; in another the ataxia is very like that of tabes dorsalis; while in still others the ataxia is of a mixed or combined form. Generally speaking, the patient staggers towards the side in which the disease is situated. We may remark here that lesion of the worm is not attended by any special symptoms that do not occur from involvement of other parts of the cerebellum; thus the theory espoused by Nothnagel, that involvement of the worm really causes symptoms ordinarily attributed to disease of the cerebellum, receives no support from clinico-pathological evidence. As a matter of fact, from what we have said concerning the connections of the worm, it follows that symptoms of disease of this part are essentially those which are characteristic of disease of the cerebellar hemisphere, except that possibly the ataxia and incoördination are greater in the cephalic extremity than with disease of the hemispheres alone.

Deficiency of muscular tone, amounting in some cases to almost absolute loss of contractility, often referred to as paralysis, occurring in the extremities, is a moderately constant symptom of cerebellar disease; whether it occurs more commonly on the same side as the lesion or whether it is developed with equal intensity on both sides it is not possible to say with certainty.

The deep-seated reflexes are sometimes normal, sometimes exaggerated, sometimes diminished and lost; probably more often diminished than perverted in other ways. Disorder of the special senses is apparently not a common symptom of cerebellar disease, although each one of the special senses has been reported impaired, and in some cases the senses of smell and of hearing are entirely abolished, with cerebellar disease. Disturbance of the tactile sense and of the muscular sense are, judging from the reports of cases, very uncommon. When a special sense, such as that of hearing, is slowly destroyed, it indicates that the disease process has involved the vestibular root of the eighth nerve at the region of its exit. The most common manifestation of cerebellar disease in any of the cranial nerves, however, is that of nystagmus. It is not usually the typical lateral or rotatory nystagmus common to disseminated sclerosis, but consists of distinct tremors or oscillations, very similar to the tremors in other parts of the body when the cerebrum is the seat of disease. Symptoms of paralysis in the territorial distribution of the motor cranial nerve are occasionally noted with disease of the cere-

bellum. It is possible that some of these, particularly their later manifestations, are the expression of muscular asthenia similar to that common in the extremities, and that when true paralysis occurs it is probably an expression of a secondary manifestation of the cerebellar disease, namely, increased intracranial pressure, and not due to disease of the cerebellum itself. The same may be said of the other incidental symptoms which were mentioned when the cerebellar symptoms were enumerated, such as polyuria, glycosuria, and the like. The significance of the trophic changes that sometimes occur is obscure.

Developmentally the cerebellum is of the same origin as the cerebrum; its vascular supply is from the same sources and it is probable that it is liable to the same diseases. In fact we have described abscess, tumor, disseminated sclerosis, and atrophy of the cerebellum when treating of those diseases as they occur in the cerebrum.

The only disease of the cerebellum of which we shall attempt a separate description is hereditary cerebellar ataxia. The functions of the cerebellum are as yet, in large part, a closed book, although the labors of Ferrier, of Luciani, of Turner, and of other investigators are seemingly in a direction to disclose them.

The general symptoms of involvement of the cerebellum are very often dubious and uncertain. The principal manifestations pointing to involvement of this portion of the encephalon are more clearly manifest when the vermis is the seat of disease or encroachment. Other symptoms pointing to disease of the cerebellum with less certainty than those that have been mentioned are: nystagmus, scanning speech, intention tremor, profound asthenia of the muscles of the same side of the body, and loss of knee jerks. Lesions of the hemispheres alone have been frequently observed without the occurrence of any so-called "cerebellar" symptoms. Lesion of the middle cerebellar peduncle causes inability to maintain equilibrium, vertigo, and peculiar compulsory, rotatory movements, such as turning on the long axis, etc.

The cerebellum would seem to have a definite function in regulating continuous and tonic muscular contraction, but just how it does this is yet unknown.

Hereditary Cerebellar Ataxia.

The name hereditary cerebellar ataxia has been given to a complex of symptoms occurring in the early years of life, always on a familiary or hereditary basis, and characterized principally by ataxia of a cerebellar nature. It is included under diseases of the cerebellum in this treatise because its exact nosological place is not yet

definitely determined. There can be no doubt that the cerebellum is the principal seat of lesion, but in all probability the disease process is by no means confined to this part of the central nervous system. When we say that it forms one of the familiary diseases, and that it belongs in the class of defective protal development, we have said all concerning its etiology and pathogenesis that can truthfully be said. The name hereditary cerebellar ataxia was given to it by Marie because it indicated the important clinical manifestation and suggested also the *sine qua non* of its genesis, heredity.

History.

The first cases carefully observed were by Frazer in 1880, but it was not until eleven years later, when Nonne described three cases, all of the same family, that the disease took a definite place in medical literature. Before that time the disease had been considered an anomalous form of hereditary spinal ataxia, Friedreich's disease. In 1892 Sanger Brown published a paper on family ataxia which included the report of no less than six cases of this disease. Appended to this paper were valuable remarks by Ormerod and Bernhardt, who both emphasized the necessity of considering these cases apart from Friedreich's disease. Since that time reports of cases have been made by Klippel and Durante, by Brissaud and Londe, by Londe, by Collins, and by Menzel.

Symptoms.

The disease usually shows itself first in late childhood and early adult life by some disturbance of gait. This in the beginning may be nothing more than clumsiness in the execution of complex coördinated movements, such as dancing, climbing, and other agile accomplishments. Pain is neither an early nor a usual symptom. As the disease progresses, however, the gait becomes uncertain and reeling, and when it is fully developed it is the typical gait of one profoundly inebriated. The patient walks with the body bent forward, the head thrown back, and the feet wide apart. The patient does not usually watch his feet, and on standing the Romberg symptom is not commonly made out. There are incoördination and loss of dexterity in the upper extremities and frequently choreiform movements which are exaggerated on voluntary effort. In some cases there are oscillatory or jerky movements of the head and less often of the trunk. Cases in the advanced stages of the disease not infrequently show the phenomena known as titubation both on standing and on sitting;

that is, inability to preserve a constant perpendicular of the body. When the patient lies down the ataxia becomes very much less manifest, oftentimes completely disappears, but the incoördination persists. The face shows exaggerated action or over-contraction of the mimetic muscles on speaking, and the speech, which is disordered from the beginning, is hesitating, abrupt, explosive, ataxic, and defective. The eyeballs have a short, jerky, mild nystagmiform movement, but true nystagmus does not often occur. Deficiency in the action of the external recti muscles has been noted by Nonne and by the writer. Optic atrophy and progressive choroiditis seem to occur in a considerable proportion of the cases, and contraction of the visual fields and diminution of visual acuteness are often early symptoms. The functions of other cranial nerves may also be impaired. Myotatic irritability is preserved, often increased all over the body; reflexes, such as the knee jerk and elbow jerk, are exaggerated, and oftentimes there is ankle clonus. Deformities of the extremities and spine do not form a part of the disease, although in an otherwise typical case reported by the writer a moderate degree of clubfoot was present. The patient makes no complaint save occasionally of headache, vertigo, or vomiting, and more rarely of cramps in the extremities and pain. Mental shortcomings, varying from slight psychical disturbances through varying degrees of idiocy up to complete dementia, are very common. The sphincters are usually intact until the disease is well advanced; then they become seriously affected. Disturbances of sensation have not been heretofore noticed except in one case. The disease is essentially a progressive one and results early in complete incapacity of the patient for any of the customary duties of life. The patient eventually becomes completely bedridden, not from muscular weakness, but from sheer inability to utilize his strength, and sinks into a state of marasmus which death terminates after a few years.

Etiology.

The disease seems to develop in the period extending from late youth up to maturity, although first evidences of it may show themselves in early infancy, as in the case I have elsewhere recorded. Of the cases so far observed a larger number occurred in males than in females. This is in keeping with the fact which is generally conceded, that in all of the familiary and hereditary diseases of the nervous system, males are more frequently affected than females. A history of antecedent or coincident tuberculosis has been observed in the immediate family. Factors which may be considered incidental in

the etiology, such as fright, trauma, and the occurrence of infectious diseases, have been noted. Parental syphilis does not seem to play any part. The entailment of neurotic inebriety is sometimes thought to have been made out. The disease is essentially a family one. No cases have been heretofore recorded in which familiary manifestations were lacking. It is not hereditary in the strict sense of the term, insomuch as one is not always able to trace it in the ancestors.

Pathological Anatomy.

Autopsies have been made on cases of hereditary cerebellar ataxia by Frazer and by Nonne and on an atypical form of the disease by Menzel. The principal pathological lesion was confined to the cerebellum. In the case of the first-named author the weight of the cerebellum was reduced by one-half, and in Nonne's case one-third. The atrophy of this organ seems to be general, although in Menzel's case the upper part was in a much more advanced state of atrophy than the lower, and the vermis was comparatively intact. It is needless to say that the atrophy may reveal itself only on microscopical examination. The membranes covering the cerebellum have been found normal except in the case of Frazer, in which the pia mater was the seat of innumerable little cysts.

Microscopically the changes in the cerebellum are seen to be a reduction in number and atrophy of the cells of Purkinje. Sclerosis in the strict sense of the term has not been described. In one of Nonne's cases the pathological changes were not confined to the cerebellum; there was a general atrophy of all the nervous system. It is more than probable that this is a keynote in the explanation of the genesis of the disease and that the various anomalous forms of hereditary cerebellar ataxia that have been described, such as the cases of Seeligmüller, Erb, Menzel, and some recent cases of Nonne, anomalous and transitional forms of the disease, will probably be found to vary in their clinical manifestations as the different interconnected and separate portions of the nervous system are the prominent seats of the protal defect.

Diagnosis.

This disease must be diagnosed from hereditary spinal ataxia, Friedreich's disease; from disseminated sclerosis; from infantile cerebral palsies of a familiary type; from chronic internal hydrocephalus; and possibly from simple atrophy of the cerebellum. The essential factors in differentiating it from the first-named disease are, first, the preserved or increased myotatic irritability, knee jerks, ankle clonus, elbow jerks. In Friedreich's disease the reflexes are dimin-

ished or absent, and a case in which ankle clonus was an accompaniment has never been described. Second, the absence of true nystagmus. Third, the striking mental defect. The early onset of the hereditary cerebellar ataxia, the absence of sensory symptoms, the intensity of incoördination in the upper extremities, and the titubation are uncommon concomitants of Friedreich's disease.

It is often extremely difficult to diagnose it from multiple or disseminated sclerosis. In fact this differential diagnosis cannot always be made because islets of sclerosis developing in the cerebellum may add that to the clinical picture of the latter disease which makes it simulate exactly hereditary cerebellar ataxia. The points to be considered in making the differential diagnosis are: First, multiple sclerosis is neither a familiary nor an hereditary disease. The clinical course of mulitple sclerosis is characterized by periods of great amelioration followed sooner or later by profound aggravation, while hereditary cerebellar ataxia is always slowly progressive. Optic atrophy particularly limited to the temporal sides of the optic discs and true nystagmus are common objective manifestations of multiple insular sclerosis. Sensory shortcomings, vesical and rectal incontinence, are also common in the latter disease, but they do not form an essential part of the former. Paraplegia, which is so often the beginning of a multiple sclerosis, is not characteristic of the advent of hereditary cerebellar ataxia nor does it usually occur as such during the course of the disease.

Cerebral diplegia of a family type such as we have referred to in the chapter on infantile cerebral palsy may closely resemble the symptoms of hereditary cerebellar ataxia. But the time of occurrence of the former, the attributable cause, the predominance of symptoms indicating cerebral defect, and the course of the disease must make the diagnosis.

Treatment.

The treatment of hereditary cerebellar ataxia differs very little from the treatment of Friedreich's disease and tabes dorsalis. The most important factors in the treatment are the maintenance of nutrition and the utilization of some such educational method as the purposive movements suggested by Fraenkel. Medicinally tonics and restoratives, and massage and hydrotherapy should be employed.

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DISEASES OF THE OBLONGATA.

Before disease of the oblongata can be thoroughly understood, it is necessary to have a comprehensive grasp of the structure, as well as of the individual and general functions of this part of the nervous system. The oblongata, medulla oblongata, or spinal bulb (*verlängertes Mark*, bulbus rhachidicus), is that portion of the central nervous system which extends from the posterior border of the pons to the lower end of the pyramidal decussation. Its upper end reaches to the clivus of the sphenoid bone; its lower to the upper border of the atlas. It is about one inch long, three-fourths of an inch wide, two-thirds of an inch thick. This small area is the highway of communication, and practically the only one between the brain and the body, the area which incoming stimuli must traverse in order to enter into consciousness, the area through which outgoing impulses must pass in order to become manifest—an area which is the seat of the only absolutely essential automatic vital functions.

Anatomy.

An organ which has such varied and complex functions must, of necessity, be complicated in structure. Its general plan of architecture is essentially that of the spinal cord, with the addition of other structures and relationships. Like the spinal cord, the white matter is located peripherally, the gray matter centrally. The anterior and posterior fissures of the spinal cord are continued in the oblongata; the former is more pronounced than the latter. The anterolateral sulcus of the cord is represented in the oblongata at the points of exit of the roots of the hypoglossal nerve, while the exit of the roots of the glossopharyngeal, vagus, and spinal accessory indicate the position of the posterolateral sulcus. On the dorsal surface, between the pyramids and the latter, are two conspicuous, rounded, oblong elevations—the olivary bodies. The transformation from spinal medulla to oblongate medulla is most easily comprehended by tracing the individual structures of the former into the latter, and then by considering the latter as a unity.

The purely motor tracts of the cord, the anterior pyramidal and the crossed pyramidal, are represented in the oblongata by the pyramids, and by the decussation of the pyramids. This decussation, which occurs in the lower third of the oblongata in the anterior area, contains other fibres than those represented in the cord, because the surface area of the pyramids above the decussation is greater than the combined surface area of the pyramids below the decussation. The destiny of the fibres which give this greater area is unknown.

The posterior columns of the spinal cord are represented by the posterior columns of the medulla oblongata. The direct cerebellar tracts are continued into the external segments of the restiform bodies. The remainder of the lateral column passes apparently into the lateral area of the oblongata. At the lower end of the olive it becomes obscured by the latter and by the passing of the internal arcuate fibres.

The gray matter of the oblongata, in its transition from the cord, undergoes more striking changes than the white. This is due to the decussation of the pyramids, which completely severs the anterior horns, and to the gradual approach of the central canal to the posterior border and its expansion into the fourth ventricle. Both of these cause such changes in the appearance of the gray matter that it no longer resembles the two crescents united at their points of greatest convexity, as it does in the cord. The crossing of one lateral pyramidal tract to the other side completely severs the anterior horn, and then the gathering together of the pyramidal bundles on the opposite side of the anterior median fissure pushes the decapitated portion of gray matter further laterally. The base of the anterior horn lies close to the central canal. The gray matter of the posterior horns is pushed laterally in a very similar way by, first, the expansion of the central canal into the fourth ventricle; and second, the increase in size of the posterior columns, the funiculi graciles et cuneati and their nuclei. The separated anterior horn and the posterior horn thus approach each other in the lateral area, and internally in the oblongata. This area of the oblongata is traversed by fibres running in a lateral direction, principally the internal arcuate fibres *en route* to their interolivary decussation. On account of this intersection, this portion of the lateral area has a reticulated appearance, and is therefore known as the reticular formation, the motor field of the tegmentum. The reticular formation is thus made up of gray matter with its ganglion cells, intersected by bundles of white fibres, which, taken together, is known as the nucleus reticularis tegmenti.

The lateral areas of the oblongata contain, just behind the pyra-

mids, three masses of glia tissue, resembling a puckered leaf, in which are embedded numerous small ganglion cells, known respectively as the olive, the inner and the posterior olive. Between the olive and nucleus reticularis tegmenti is a small field of longitudinal fibres, which is believed to be made up principally of brain-olive fibres (central tegmental tract). As far brainward as beyond the superior pyramidal decussation a portion of gray matter, rich in nerve cells, many of them of large size, may be seen projecting laterally just beyond the olive. It is called the nucleus lateralis, and may be regarded as the upward prolongation of the lateral horn of the cord. The posterior horns are pushed laterally and nearer the surface. The base of the posterior horns becomes constricted to form the cervix, the constricted mass or caput enlarges, and finally becomes severed by the passage of transverse and longitudinal bundles, in much the same way as do the anterior horns.

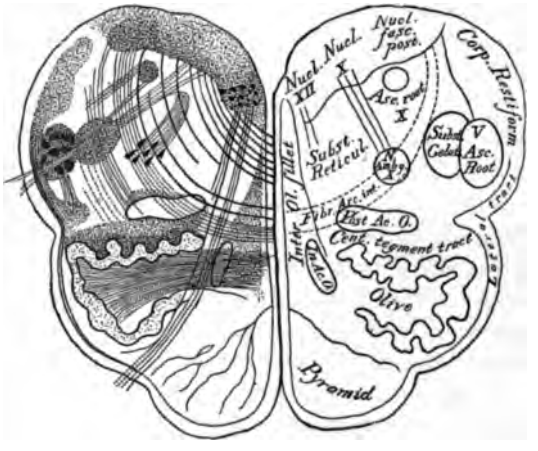

Fig. 19.—Cross Section of the Oblongata.

This decapitated posterior horn, lying in the posterior area of the oblongata laterally to the cuneate fascicle, forms the funiculus of Rolando; and higher up it causes such a prominence on the surface that it is called the tubercle of Rolando. Portions of the gray matter of the posterior horns, projecting into the funiculus gracilis and cuneatus, form the nuclei of the latter. It is around these nuclei that the greater portion of the peripheral sensory neuraxons making up the posterior columns arborize, and from these nuclei arise the internal arcuate fibres, which, reaching the middle line in an arciform manner, have their decussation just above the pyramidal decussation, and then (fillet decussation) pass towards the brain, as the fillet or lemniscus. A cross section of the oblongata (Fig. 19) above the pyramidal decussation shows on the periphery anteriorly the great pyramidal masses; laterally and toward the posterior surface, the restiform bodies, which constitute the inferior cerebellar peduncles. These latter have been divided into an external and an internal portion, but for our purpose they may be considered as one body,

made up of the direct (dorsolateral) cerebellar tract, fibres from the province of the olives and the formatio reticularis by way of the anterior arcuate fibres, and fibres from the posterior columns, which pass to the restiform bodies by way of the external arcuate fibres.

The arrangement of the cranial nerve nuclei is shown graphically by Fig. 19, a cross section of the oblongata at the junction of its upper and middle third, and by Fig. 20, a longitudinal section through the entire length of the medulla oblongata, and which is to be considered transparent.

In the inferior half of the oblongata the nuclear region or area is composed principally of the nucleus of the hypoglossal, the motor and sensory nuclei of the glossopharyngeal and vagus, the solitary

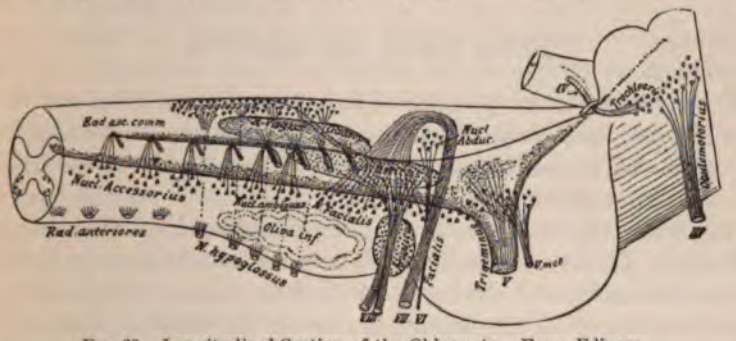

FIG. 20.—Longitudinal Section of the Oblongata. From Edinger.

fascicle or bundle which is considered the ascending root of the vagus-glossopharyngeal, the remains of the nuclei of the posterior columns, the portion of the substantia gelatinosa which comes from the posterior horns, and the adjacent so-called ascending (really descending) root of the fifth nerve. Sections of the oblongata further brainwards reveal other nuclei, the most important of which are the nucleus of the facial; the nuclei of the auditory, dorsal and ventral; Deiter's nucleus, formerly erroneously supposed to be the external auditory nucleus; and the nucleus ambiguus, which lies in the space between the superior accessory olive and the substantia gelatinosa, traversed by some of the internal arcuate fibres, and looked upon as the motor vagus-glossopharyngeal nucleus. The nuclei in the oblongata have, relatively, the same arrangement as those in the spinal cord, the motor nerves lying mesally, the sensory towards the substantia gelatinosa. The origin and termination of the motor and sensory neurons in their respective nuclei in the oblongata are similar to those in the cord. According to Held, the sensory vagus and glossopharyngeal terminate in the ala cinerea, the function of which is to contribute to the preservation of equilibrium, and are distributed along the entire floor

and lateral wall of the fourth ventricle; while the acoustic division of the eighth nerve, the real auditory nerve, terminates in the anterior acoustic nucleus and acoustic tubercle. The branches of the descending portion of the root fibres of the glossopharyngeal and vagus form the solitary bundle; the descending branches of the trigeminal roots, the so-called ascending root of the trigeminal. The descending branches of the vestibular nerve pass in the so-called inner division of the inferior cerebellar peduncle. The ascending root fibres are formed gradually by the fine-ending collaterals. In addition to these vestibular branches in the inner portion of the cerebellar peduncles, there are fibres from the trigeminus, the vagus, and the glossopharyngeal.

Blood Supply and Functions.

For a ready comprehension of the clinical manifestations of vascular lesions, such as hemorrhage, thrombosis, and embolism, in the oblongata and pons, it is necessary to understand the blood supply of this portion of the nervous system.

The following brief retrospect is based upon the well-known work of Duret. The blood supply of the oblongata and pons is furnished by the vertebrals, and the product of their union, the basilar. The immediate vessels of the oblongata may be classified as lateral, radicular for the nerve roots, median, nuclear for the nerve nuclei, and the arteries for the olives, pyramids, and the remainder of the oblongata. Each radicular artery divides into an ascending and a descending branch; the first passes along the root fibres to the nuclei of their origin, the second accompanies the root fibres towards the periphery.

The median arteries may be divided from below upwards into four groups: 1. The most deeply situated arises from the anterior spinal artery, artères bulbaires. 2. Arteries which arise from the lower division of the basilar artery at the inferior border of the pons, artères sous-protubérantilles. 3. Arteries which arise directly from the basilar, artères medioprotubérantilles. 4. Arteries which arise at the upper point of division of the basilar artery, artères sus-protubérantilles.

These median arteries are of very small calibre, and are of the terminal sort, as no anastomoses can be seen. They pass from the ventral surface upwards in the raphé towards the floor of the fourth ventricle and aqueduct of Sylvius, where they terminate around the nerve cells there grouped. The ramifications of their capillary vessels are adjacent and in the same environs as those of the ascending branches.

The arteries of the last group are: (*a*) Those for the ventral portion of the oblongata, the pyramids, and olives, which arise in part directly from the vertebral artery, and in part from the radicular arteries and the anterior spinal. Generally two or three branches can be seen to pass, with the roots of the hypoglossal, through the hilus of the olive. (*b*) The vascular supply for the lateral portion of the oblongata, the restiform body, is derived from the inferior cerebellar artery. The vascular supply for the dorsal portion, which is evidently given through the inferior tela choroidea and the choroid plexus, is also from the inferior cerebellar artery.

The functions of the oblongata are practically the same as those of the spinal cord, conductive, reflexive, and trophic, the latter, of course, including vasomotor.

The conducting pathways were referred to when considering the anatomy. The most important reflex centres are the centres for closure of the eyelids, for sneezing, for coughing, in connection with the centre for closure of the glottis, and for the movements of chewing, sucking, swallowing, vomiting, and regurgitation.

In addition to these purely reflex centres, there are centres, partly reflex, apparently automatic, such as the respiratory centre in the posterior portion of the calamus scriptorius, a centre for the regulation of the heart, a centre for slowing and for increasing the number of beats, and a centre for inhibitory control of spinal reflexes—a centre known as Setschenow's centre. The vasotonus centres are in the gray matter of the floor of the fourth ventricle; that part which presides over the vasomotor tone of the kidneys is believed to be adjacent to the vagus nucleus, and in juxtaposition to the point where Claude Bernard found that a puncture caused sugar to appear in the urine, probably by injury of the vasomotor centre of the liver.

The general vasomotor centre in the oblongata is the one to which the spinal vasomotor centres are subsidiary. They (for it is probable that there is one for dilatation as well as for constriction of the vessels) lie along the floor of the fourth ventricle and in the immediate environment of the common glossopharyngeal-vagus-accessorius nucleus. In disease of the latter, such as bulbar palsy, a perversion of vasomotor function is frequently apparent.

From clinical observation we are led to the belief that there exist also in the oblongata secretory centres, salivary and perspiratory. As will be discussed hereafter, glosso-labio-laryngeal palsy is often accompanied by excessive production of saliva, less often by hyperidrosis. The intimate relationship of these manifestations with the vasomotor system, and their possible dependence upon the former, cannot be touched upon here.

Classification of Bulbar Diseases.

It is only within the present decade that diseases of the oblongata, apart from chronic bulbar palsy, Duchenne's paralysis, have been considered systematically in the text-books. The time has come, I believe, when this should no longer be so, and here the affections of the oblongata will be discussed, in point both of precedence and space, according to their relative frequency and importance. The diseases which affect the oblongata may, for convenience' sake, be considered as: 1. Primary degenerative lesions, labio-glosso-laryngeal paralysis, chronic progressive bulbar paralysis. 2. Primary vascular lesions: (*a*) Acute inflammatory bulbar paralysis; (*b*) bulbar hemorrhage; (*c*) thrombosis and embolism. 3. Bulbar neuritis, associated neuritis of the bulbar nerves. 4. Infantile bulbar paralysis: (*a*) Familiary, (*b*) hereditary. 5. Secondary degenerative lesions of the bulb: (*a*) in amyotrophic lateral sclerosis; (*b*) in syringomyelia; (*c*) in tabes and multiple sclerosis. 6. Pseudobulbar paralysis (cerebral). 7. Tumors of the oblongata, including gummata. 8. Bulbar paralysis without anatomical foundation, asthenic bulbar paralysis.

Chronic Progressive Bulbar Paralysis.

(Labio-glosso-laryngeal Paralysis.)

This is a well-defined combination of symptoms, associated with unvarying anatomical changes, which has been recognized only during the latter half of the nineteenth century. Clinically, it consists, as the name implies, of a paralysis of the lips, tongue, and larynx, causing a destruction of some or all of the functions of these parts, and associated with more or less atrophy, particularly of the lips and tongue.

It has received different names from different writers. It is called by Leyden, progressive atrophic bulbar paralysis; by Kussmaul, paralysis of the bulbar nuclei; by Wernicke, chronic inferior polioencephalitis, in contradistinction to an entirely different syndrome, first described by this author, known as superior polioencephalitis; by Wachsmuth, progressive bulbar palsy. All of these names are inferior to the name by which the disease is generally known, as they aim to indicate the morbid anatomy of the disease or some feature of it, while they neglect to convey the essential clinical features. The first aim of a definition should be the latter, and if it serves this

purpose clearly it is to be preferred to an anatomical or eponymic one.

History.

In 1860 Duchenne, the renowned clinician of Boulogne, described an example of this disease under the title of "progressive paralysis of the tongue, of the soft palate, and of the lips." For a long time thereafter, and occasionally even to the present date, the disease was referred to as Duchenne's paralysis. Like many other diseases for whose recognition we are indebted to the phenomenal clinical insight of this physician, the affection first came prominently before the medical profession after being discussed and described by another illustrious French physician, Trousseau. It is remarkable that Trousseau had met with and made notes of a case of this disease in 1841, twenty years before. This observation, however, remained lost to the profession until the time Duchenne's descriptions were commented on. It is not uncommon to find it stated that Duchenne was the first to describe a case of this disease, but this is an error. Examples were described in 1859 by Dumesnil, of Rouen, and even as far back as 1825 by Robinson. Nevertheless this in no way detracts from the credit of Duchenne, as we may be quite certain that he was ignorant of these contributions, and indeed it may be said that they were apparently lost in the literature, and had failed to call attention to the existence of the disease. One of Duchenne's first contentions was that the disease was something entirely different from progressive muscular atrophy, and that it was an instance of paralysis, without atrophy. Whether it was a lesion of the central nervous system or some neurosis, he was unwilling to say.

During the decade following Duchenne's observation, cases were noted by other clinicians, but the dependence of the symptoms upon destruction of groups of ganglion cells or nuclei in the medulla oblongata was not recognized until later, though Clarke had, in 1867, discovered in a case of labio-glosso-laryngeal paralysis associated with atrophy of the tongue that the nucleus of the hypoglossus and the adjoining nuclei were atrophic. This association was not looked upon as anything more than coincidental.

It was not until 1869, when Charcot and Joffroy, in France, and Leyden, in Germany, began their investigations into the pathology of this affection, that the changes in the oblongata were considered responsible for the well-defined symptom complex, although the central seat of the disease and its connection with the oblongata had been postulated by Baerwinkel, of Leipsic, in 1860, by Wachsmuth, of Dorpat, in 1864, and by Schultz, of Vienna, in the same year.

The microscopical examination made by Kussmaul and Maier, in 1873, corroborated the views of Charcot and Joffroy, and of Leyden, and went far towards extending the real knowledge of the disease.

Some discussion has arisen as to whether the honor of having interpreted the anatomical accompaniments of the disease should be accorded to Charcot and his pupils, or to Leyden, but the facts seem to be as stated by Brissaud, that in 1869 Leyden made a communication on this affection to the congress at Innspruck, which did not contain any allusion to the rôle which the motor cells of the bulbar nuclei play in the physiological pathology of this disease. Later, however, this author pointed out in the most lucid manner the dependence of the symptoms individually and collectively upon the changes demonstrable in these nuclei, and the nerves issuing from them.

The neuropathic theory of bulbar paralysis found its stanchest opponent in Friedreich, who contended that the changes in the nerve cells were secondary to those in the muscles, the same as in progressive muscular atrophy. Time has shown that he was right in his analogy, but wrong in his premises.

Much profitless discussion has been indulged in to explain the absence of muscular wasting in some cases and its occurrence in others. In fact, the endeavor to solve this enigma prompted the many diverse views of the anatomical nature of the disease. Since that time a great number of contributions summarizing the knowledge of the subject, or pointing out some new clinical or pathological feature, have appeared.

Our understanding of the disease has been furthered particularly, however, by the study of chronic anterior poliomyelitis, of amyotrophic lateral sclerosis, and of syringomyelia, and also by distinguishing from this form of bulbar palsy such diseases as bulbar neuritis, pseudobulbar palsy, bulbar thrombosis, and embolism, polioencephalitis superior, and asthenic bulbar paralysis, each one of which gives rise to a complex of symptoms so very like the form that we are considering that in some cases it is impossible to make the differential diagnosis *intra vitam*. Each one of these simulating conditions will be considered in detail later on. In fact, it should be said as emphatically as possible, that progressive bulbar paralysis in its strictest sense, the sense in which we here consider it, is a very rare disease; rarer, in fact, than it was formerly considered, and this partly because we are now in position to diagnose it from the diseases just enumerated.

Etiology.

The actual causes of this disease, like those of almost all the degenerative diseases of the nervous system, are unknown. That which will cause a group of nuclear cells in an apparently normal individual to take on degenerative changes which progress gradually to complete destruction we may postulate, but we cannot prove. A theory has been advanced that degenerative systemic diseases occur in parts that have been excessively used; that is, that degenerative changes are the natural successors of exhaustion. The force of this hypothesis is dependent largely on coincidences in which degeneration of tracts or nuclei has followed the exhaustive use of parts that they supply. Progressive bulbar paralysis is one of the diseases that has been used as an example to support this theory, because cases have been observed in glass-blowers, buglers, cornet players, and others who put their lips, tongue and throat muscles to prolonged and excessive use. It is unfortunate that there are no records of autopsy in any case in which such causative factors were apparently operative; since the separation of a form of bulbar paralysis without anatomical foundation has been made, the suspicion naturally arises that such cases were examples of the latter condition, which, as will be pointed out later, is either a toxic or an exhaustion neurosis, or both. Progressive bulbar paralysis is a rare disease at any age, and particularly so in the young, less so in the senile. In the former it rarely occurs, and then only as a familiary or hereditary form, or as an upward extension of a high poliomyelitis, thus constituting a bulbar myelitis as in a case reported by Hoppe-Seyler. There are a few cases occurring in childhood on record in which no familiary or hereditary transmission can be traced, as a case observed by Hoffmann at eleven years, one by Remak at eight; but it may be safely said that true degenerative paralysis occurring in infancy is the rarest of all nervous diseases. The disease occurs about one-third more frequently in males than in females, and the cases observed in females develop at relatively a more advanced age.

Factors which are often held responsible as causative of degeneration of the nervous system in other parts of the body, such as syphilis, rheumatism, and gout, cannot be posited as etiological factors in this disease, it being rare to find that the poison of these diseases had ever found tenancy in the system, nor can it be said that the disease is closely associated with degeneration of blood-vessels aside from the fact that it commonly occurs at an epoch when vascular changes usually take place.

The exciting causes that may be mentioned are, first and most important, over-exertion, particularly of the mouth and vocal apparatus, fright and anxiety, enervating habits, exposure to cold, and all forms of depraving influences. Theoretically, it is considered that toxic factors may be operative in some cases, but the only proof of such that can be advanced is one of analogy. In a very few cases, however, it has been observed that the disease occurred after lead poisoning, diphtheria, and influenza. In one of these reported by Gowers, the patient was in advanced life, and the bulbar palsy may have been merely a coincidence, not a consequence. In considering the cases that have been reported following diphtheria, it must be borne in mind that many of them were reported at a time when the symptom complex now described under bulbar neuritis was unknown. I am inclined to believe that the case reported by Stadthagen, which followed diphtheria, was one of associated neuritis of the bulbar nerves, as such sequelæ of diffuse polyneuritis as atrophy and contracture of the extremities developed. A few cases of degenerative bulbar palsy are secondary to an acute, vascular bulbar palsy. Lastly there remains to be mentioned a certain number of cases that supervene on and accompany other degenerative diseases, such as progressive muscular atrophy and amyotrophic lateral sclerosis, syringomyelia, tabes dorsalis, and multiple insular sclerosis. In the first two the degenerative process would seem to be but an extension upwards, with consequent involvement of groups of cells which are architecturally a direct continuation of those involved in the cord. In a similar way the acute form has been seen superimposed on anterior poliomyelitis. The inter-relationship between these diseases and degenerative bulbar paralysis will be considered under the respective diseases.

Symptoms.

In many instances it is difficult for the patient to fix accurately the onset of the disease, so slow and insidious is its beginning. As a rule patients will say that for a time they have noticed that fatigue is more easily induced; that they are irritable, nervous, and easily upset; that they are possessed of undefinable fears; in short, the symptoms are more or less severe general neurasthenic symptoms. Then, after some excitement, severe strain, or exhausting influence they remark either some slight difficulty in swallowing or a lack of distinctness in speech, which is apparently dependent upon defective control of the lips and tongue, manifesting itself by loss of mobility or dexterity in these parts. This is associated with and often preceded by a feeling of trembling or twitching in the lips,

muscles of the face, and of the neck. Often these quick fibrillary twitchings can be seen early in the disease by the physician. With these is associated what the patient says is neither a pain nor a soreness, a sort of a painful stiffness of the muscles that support the head. Often a quickly induced feeling of exhaustion in the upper extremities and the face, a real amyosthenia, is an early symptom. Prodromal symptoms, which are often present but also often absent, are palpitation of the heart, attacks of dyspnœa, and symptoms of gastric fermentation. These and other symptoms even less suggestive of disease of the oblongata may be present for several months before any of the complex of symptoms going to make up the typical clinical picture of this disease become manifest. Generally, however, they are quickly followed by disturbance of articulation, of tone production, and of swallowing, those of articulation being usually the first and most manifest. The patient notices at first when he attempts to talk rapidly, or when excited, or after he has been speaking for some time, that pronunciation becomes slurred and somewhat indistinct, and particularly that those words which are composed of several consonants and labials such as r, l, t, s, k, and vowel i, are imperfectly formed and enunciated. As the intensity of the disease increases, speech becomes thick and guttural, but not at all rhythmical, stammering, or explosive. The patient makes great efforts to produce articular speech, and such efforts are very exhausting. The patient has a sensation of stiffness about the mouth, and the lips show early loss of dexterity in the prehension of food, and in the finesse of movements which enter into facial expression, particularly during speaking. A loss of tonus in the same muscles allows the lower lip to fall and evert, and the upper lip to stand out from the teeth, a condition which, combined with defective innervation in the lower area of the facial nerve distribution, gives rise to a facies characteristic and nearly pathognomonic of this disease.

In many cases patients notice that unwieldiness of the tongue is an early symptom, and that frequently it gets caught between the teeth, especially if the patient is fatigued, or if he tries to masticate and swallow rapidly. It is also shown by the fact that oftentimes the finger must be used to dislodge particles or masses of food that have got between the cheeks and the teeth, work that is usually done by the tongue unconsciously and without effort. This is usually noticed before any signs of atrophy can be detected and before any perceptible limitation of extrabuccal movements of the tongue can be made out. It is a noteworthy fact, and one deserving of more universal recognition, that even after intrabuccal mobility of the tongue is almost *nil*, considerable extrabuccal lingual movements can still be

executed. Slowness of movements of the tongue is noted; and drawing the tip of the tongue along the roof of the mouth, turning it over the upper lip, compressing the tip to a point, and making a longitudinal furrow through the centre, are manipulations that soon become impossible. If the tongue be taken between the fingers it will be felt to be less resistant than usual, more or less flabby and pultaceous. Later the appearance of transverse irregularities in the tongue at rest in the buccal cavity shows the existence of well-developed atrophy. As the disease progresses the tongue becomes more and more powerless until finally it lies on the floor of the buccal cavity, broad, flat, thin, flabby, indented on the margins by the impress of the teeth, the most vigorous voluntary efforts failing to cause any perceptible movement, an occasional involuntary muscular flickering being the only evidence that it ever possessed mobility. Associated with this condition of the tongue changes of the lips develop *pari passu*. In rare instances the manifestations of paralysis are unilateral. This has been noted by Grugia and Matteucci, by Scheiber, and by Wiener. As has been said, loss of labial prehensility and agility of the lips are oftentimes early symptoms. Following these there will be inability to pucker the lips as in whistling and in kissing. I have recently seen a case in which inability of a mother to satisfactorily kiss her children was the first symptom of the disease which she noticed. The same causes which are operative in preventing puckering of the tongue when within the mouth and when protruded are responsible for this inability to pucker the lips, as the one action subserves the other. With these changes in the lips and tongue articulation becomes much more defective, and this, combined with the changes in the voice which will be described later, reduces the patient to a condition in which he is unable to communicate his thoughts by spoken words. Long before this condition of complete inarticulation known as anarthria develops, the patient will complain that the efforts necessary to speech are very exhausting, and it is noticeable that such efforts materially increase the dysarthria of the initial stages of the disease.

In the large proportion of cases drooling, the flowing of saliva from the mouth, is an early and continuous symptom. Whether or not there is constantly hypersecretion of saliva is not definitely settled. It is probable that often the amount of secretion is not only in excess, but greatly so. This hypersecretion, which may amount to from one to two pints in twenty-four hours, is due in part, if not entirely, to mechanical reflex stimulation of the buccal mucous membrane. The lips having lost their tonicity the aperture of the mouth remains open, and not only do the irritating substances of the atmosphere come in contact with the mucous membrane but the air itself

excites secretion. In some instances the amount of saliva secreted has been so great that it has been thought it might be due to irritation of the chorda tympani and petrosal nerves, which convey vasodilator and true secretory impulses to the submaxillary and parotid glands respectively. In the later stages of the disease there is apparently a greater amount of saliva than exists really, for, on account of the difficulty of swallowing, this secretion, which naturally passes into the stomach, collects in the mouth, and the paresis of the lips and tongue allows it to dribble.

To collect the saliva these patients almost always hold a handkerchief before the mouth, an attitude which Charcot's verbal and pictorial description made famous. Some patients with this malady complain, not of drooling or hypersecretion of saliva, but of annoying dryness of the mouth, which is present not only on awakening, when it may be accounted for by the fact that these patients sleep with open mouths, but in the daytime as well. When the latter exists, sense of taste, especially acuteness of the sense, suffers proportionately, because the presence of saliva is contributory to the latter.

In health, when the sound "ah" is uttered, or when the uvula or soft palate is touched, there results a quick elevation of the soft palate and its free attachments to which the name palatal reflex is given. In glosso-labio-laryngeal paralysis this reflex is absent and it frequently fails early. This failure when complete is not only responsible in part for the disagreeable nasal twang which the voice early betrays, but involves serious entailment when it becomes more complete by allowing fluids to regurgitate through the nose on attempting to swallow, and later when it is associated with defective epiglottis action by contributing to the ease with which particles of food pass into the glottis. Facility of deglutition suffers early in the disease. At first, like so many other of the beginning symptoms, it is noticed only when the patient is fatigued, hurried, or excited; then it is noticed that after swallowing a few times in rapid succession, the swallowing reflex does not occur involuntarily, but that the substances to be swallowed must be forced down, and even the ability to do this comes only after resting for a time. At first solids and particularly semisolid substances are swallowed with more ease and readiness than liquids, but as the disease progresses a stage is reached in which only liquids can be taken. Eventually this stage is succeeded by a period when the only way of getting alimentation into the stomach is by means of a tube. Some ability to swallow solid food is often preserved long after the patient voluntarily relinquishes it because of the fact that frequently on taking the first mouthful a

small particle will escape into the glottis and cause the most distressing and agonizing attacks of spasmodic coughing, the termination of which finds the patient in a most exhausted condition.

The changes in the voice are so distinctive that they are frequently referred to as bulbar. The first manifestation is a distinct nasal twang, particularly noticeable when the patient makes efforts to speak loud and to raise the voice. Associated with this is a loss of the production of high notes as in efforts of singing. Like the symptoms mentioned heretofore, the laryngeal symptoms betray themselves first when the patient is fatigued or on more or less sustained effort. Prolonged periods of rest of the voice in the beginning of the disease are followed by increased capability of voice production, which, however, is soon exhausted. As the disease progresses vocalization becomes more and more toneless, and this, associated with defective letter and word formation, puts the patient beyond the capability of verbal communication. Coincidently with loss of tone of the voice absence of the normal sound or tone which accompanies coughing and sneezing occurs. These develop long before the expulsive power of the diaphragm and expiratory muscles is materially diminished.

Associated with the development of local symptoms in degenerative bulbar palsy there are in individual cases variable general symptoms. These are stiffness and pain in the neck and shoulders, occurring in connection with twitchings in the musculature of these parts; feeling of extreme weight and heaviness of the upper extremities and exhausted sensation after exertion, particularly exertion with raised hands, such as is required in taking food, dressing the hair, etc. A similar condition of amyosthenia develops in the lower extremities. The patients may complain even from the beginning of weakness and a feeling of quivering and unsteadiness in the lower extremities. After a time they complain that although walking on a level surface is easy, the slightest elevation, such as stepping from the pavement to the curb, raising the foot to go upstairs, or crossing one knee over the other, requires at first much effort and later cannot be done except by the aid of the hands. Although there is more or less progressive emaciation in addition to the atrophy of the musculature of the lips, tongue, pharynx, and larynx, the former is not commensurate with the loss of muscular strength, and the easily induced fatigue. The vegetative functions of the body are, aside from intercurrent and temporary aberrations, normally performed. The appetite is often a great deal more than can be appeased, owing to defective deglutition, and digestion is usually good. Attacks of fermentative dyspepsia, associated with annoying borborygmus, often occur, and particularly if the patient

be nervous or upset. As in all other diseases in which there is a marked depression of muscular tonus, the tongue is often coated, the breath foul, and the bowels constipated. The urine does not usually show any marked departure from normal. Although there is an area in the oblongata which if encroached upon will cause glycosuria, it is not affected in this disease, as the latter is a very unusual symptom.

The pulse is frequently rapid, 100 to 120, generally so from the beginning of the disease, and the pulse rate is easily accelerated by physical and psychical influences. The distressing attacks of cardiac dyspnœa that may occur and be early symptoms have already been spoken of. Manifestations of faulty circulation and insufficient vascular tone, such as cold extremities, mottled skin, and disturbed secretions, are usual concomitants. Attacks of palpitation and pseudoangina are very terrifying to the patient, and oftentimes the fright accompanying them serves to accentuate the severity of these symptoms. It is most uncommon for death to take place during such an attack, yet in some cases syncope has been the precursor of dissolution. These attacks of syncope do not differ from the ordinary attacks, except by their greater severity and longer duration. When the heart is auscultated during one of these attacks of cardiac oppression nothing save a confusion of the valvular sounds of the heart is heard—as Duchenne pointed out, a sound as if the heart was beating in a fluid. The function of respiration is not infrequently seriously interfered with. In the beginning of the disease the patient complains merely of getting out of breath easily, and it will be noticed that such patients take a full inspiration before beginning to speak. Compulsory and forcible expirations such as hawking, coughing, sneezing, etc., are done with progressively diminishing vigor. Normally these acts are attended with a well-marked tone, but in this disease it gradually diminishes until they become entirely toneless. If, when the patient has reached this stage, he is so unfortunate as to develop a laryngitis or bronchitis, be it ever so slight, his condition becomes a pitiable one. All the involuntary muscles of inspiration are brought into activity, and the most strenuous expiratory efforts to dislodge the accumulating secretions are abortive. The mucus accumulates in the tubes, and not being thrown off by involuntary or voluntary expulsive power of the bronchi and muscles of expiration, asphyxia rapidly develops. If the chest is auscultated during such an attack, the respiratory murmur will be feeble up to the point of complete unrecognition, and the râles naturally accompanying such conditions will be absent.

The psychical sphere and particularly the higher psychical faculties in patients affected with this disease remain practically undis-

turbed. Not infrequently these patients are emotional, emotions of depression being more common than those of elation. These emotional manifestations, it should be stated, are not accompanied by a corresponding psychical state; on the contrary, the patient may be in tears without betraying any other evidence of depression or morbidity. As in most other chronic diseases there are periods when these patients are cast down and depressed, but as a rule they are hopeful, even when their condition is distressing to look upon. In very rare instances a condition of somnolence and hebetude may develop, which, if not dependent upon anterior syphilis and if not associated with well-marked vascular degeneration, is to be looked upon as a comparatively certain forerunner of early dissolution.

In some very rare instances an increase of temperature has been noted during the course of the disease. Such a symptom posits involvement of other portions of the oblongata than those ordinarily affected in the disease. It is usually accompanied by respiratory and cardiac symptoms which bespeak a bad prognosis.

Physical Signs.

Of necessity many of the accompanying signs have been enumerated in discussing the symptoms. It can only be of advantage, however, to place succinctly before the reader the revelations of the physical examination of a patient in whom the disease is moderately well developed. The face will present the appearance already described, the upper part mobile and expressive, the lower part immobile and fixed; the ocular apertures are apparently slightly increased; there is absence of nasal respiratory movements; the lips are parted and slightly everted; the lower jaw is somewhat dropped, and there is almost always some drooling. The vision is normal, movements of the eyeballs are free, and the pupils respond promptly to light and in accommodation. In typical degenerative bulbar palsy the optic nerve is always intact. Atrophy of the nerve is reported in one or two instances in the entire literature. In very rare instances there is some evidence of paresis of the sixth nerve and consequent diplopia. I have seen one case of otherwise typical bulbar paralysis ushered in or preceded by ptosis first of one eye, then, after its disappearance, of the other, and occasional occurrence of diplopia. The senses of smell and of hearing and the faculty of equilibration are undisturbed. Occasionally manifestations of defective innervation of the motor branch of the trigeminal nerve are apparent in the lack of proper activity of the masticatory muscles. There is no defect in the sensory distribution of the fifth nerve. The deficiencies of the cervico-facial branches

of the seventh nerve have already been referred to. Sense of taste as a rule is intact; in some exceptional instances it has been found perverted or absent, especially at the tip of the tongue, but in such cases there has been found some anomaly of the sensibility of the tongue, lips, etc. Examination of the deep reflexes, the knee jerk, elbow jerk, ankle jerk, etc., show that they are diminished in the great majority of cases. In few exceptional instances the deep reflexes are found to be increased. In the latter cases this condition of the reflexes is to be explained by the supposition that the inhibitory control of the brain on the reflexes is interrupted or interfered with in the oblongata, and that there is a perversion of the function of Setchenow's reflex inhibitory centre, or that there is some primary or secondary degeneration of the primary motor neurons. Myotatic irritability is diminished not only on mechanical stimulation but on electrical as well. This is often to be seen while applying electricity, either the galvanic or faradic current, for its therapeutic effect; a stronger current will be required to produce contraction in a muscle after it has been caused to contract a number of times than at the beginning.

In the musculature wherein the primary degenerative atrophy is manifest, that is, of the lips, the tongue, the palate, pharynx, and larnyx, there is true reaction of degeneration, as the peripheral motor neuron is diseased. In the beginning of the disease, when the atrophy has not yet become pronounced, there will be a response to both the induced and the primary current, but as the disease increases and the atrophy becomes more complete, loss of response to the former will grow continually more evident. There may be reversed polar irritability to the galvanic current, but this is not at all a constant phenomenon. The superficial reflexes are generally unaltered. There is generally a well-marked fine tremor of the fingers which is accentuated by excitement and fatigue. Tremor of the tongue, and much less frequently slow vermicular or wavelike movements, are to be seen. Quick, fibrillary twitchings are sometimes seen in the lips and chin.

Laryngoscopical examination reveals the deficiencies of vocal-cord action dependent upon the amount of paralysis. In the great majority of cases almost from the beginning, there will be found some separation of the vocal cords which is due to paralysis of the abductors, the arytenoid cartilage, or the constrictors of the glottis. Towards the end of the disease, or when the manifestations of the disease are well pronounced, the laryngoscope shows, during supreme efforts at vocalization, the cords limp and widely separated, and the glottidean space seems correspondingly large on account of this laxness. In very rare cases there is paresis or paralysis of the abductors; this is shown by the opposition of the vocal cords on laryngoscopic

examination. When this condition exists, it would seem that the opposing vocal cords acted somewhat like an encroachment, inflammatory or otherwise, on the glottis and caused the distressing attacks of dyspnœa, accompanied by croupy inspirations.

Diagnosis.

If the disease has existed for some time and the symptoms are moderately well marked, the diagnosis can be made with readiness.

It would constitute an unnecessary repetition of what has been said under symptomatology to recount the pathognomonic accompaniments of this affection. There are several conditions which simulate clinically progressive labio-glosso-laryngeal palsy to such a degree that a differential diagnosis is often difficult and sometimes impossible. There are the three principal forms of pseudobulbar paralysis: cerebral, radicular, and neural, as well as the dynamic form of bulbar paralysis or bulbar paralysis without anatomical foundation, double facial paralysis, pharyngeal paralysis following acute infection such as diphtheria, tumor of the oblongata, pressure on the medulla from aneurysm, gummata, etc., progressive dystrophy of the facio-scapulo-humeral type, and disseminated sclerosis. It is necessary also to differentiate it from the bulbar syndrome sometimes developed with tabes and syringomyelia. The form of bulbar palsy that develops coincidently with or following amyotrophic lateral sclerosis differs in no wise from the disease under consideration, except that some of the purely bulbar symptoms are masked by those referable to diseases of the pyramidal projections. By pseudobulbar paralysis is meant a symptom complex simulating that of bulbar paralysis dependent upon a lesion not in the medulla oblongata. A lesion to produce such bulbar symptoms may be situated in any part of those central motor neurons which extend from the cortex of the brain to their arborization around the bulbar nuclei of the seventh, ninth, tenth, eleventh, and twelfth nerves, except the part within the oblongata and in any part of the peripheral motor neurons arising from the nuclei of these same nerves, outside the oblongata. It is unnecessary to say that the collective peripheral neurons of more than one of these nuclei must be involved to produce the symptoms of bulbar palsy.

Pseudobulbar paralysis is said to be of cerebral origin when the lesion or lesions that produce the bulbar phenomena are in the cerebral cortex, the corona radiata, the internal capsule, and the pyramidal projection to the oblongata, or at least when there is involvement of

such parts of these pathways as are occupied by the central projection of the motor cranial nerves.

As a matter of fact the lesion in true cerebral pseudobulbar palsy is generally found in the foot of the third frontal and in the ascending frontal and parietal convolutions. The next most common location is the lenticular nucleus, particularly its outer segment.

Before enumerating the points of difference in these two diseases, a word must be devoted to pseudobulbar palsy of cerebral origin. The reality of existence of the latter is now universally conceded, although even as late as a decade ago such admission was not so unanimous. Oppenheim and Siemerling in 1886 thought that it was not possible to diagnose pseudobulbar paralysis in the strict sense of the term, and that the great majority of the cases diagnosed as cerebral pseudobulbar palsy were in reality cases of associated lesion in the brain and the bulb. This statement was not accepted, and it may be said that from the time that Joffroy, and after him Lepine, showed that the typical symptom complex of bulbar palsy could be produced by well-defined lesion in different parts of the cerebrum and its ganglia, the belief in the reality of cerebral pseudobulbar paralysis has steadily gained ground, until to-day the diagnosis of the latter is so readily made that it need never be confounded with the former. It is an interesting fact that the first records that we have of cerebral pseudobulbar palsy by Magnus antedate by some years the description of progressive bulbar palsy given by Duchenne. During the past few years a considerable literature has grown up around this subject, particularly in this country and in France, and for an exhaustive consideration of the subject the reader may be referred to the theses of Leresche, Galavielle, Halipre, and Wolff. We can refer here only by name to the cases of Barlow, Jackson and Taylor, Fuller and Browning, Eisenlohr, Kirchoff, Mott, Colman, McNutt, to show how general its recognition is.

The pathology and morbid anatomy of pseudobulbar palsy of cerebral origin need not detain us, except to say that an encephalomalacia of arteriosclerotic origin is the basis of them.

That syphilis may be the cause of such a symptom complex is shown by a case related by Munzer in which it was the result of diffuse gummatous infiltration of the frontal and central convolutions.

Cerebral pseudobulbar palsy is to be differentiated from true bulbar palsy by a consideration of the principal etiological and clinical factors in its occurrence. The foregoing factors that enter into the genesis of cerebral pseudobulbar paralysis are those of cerebral apoplexy, and particularly, it would seem, those of embolism and thrombosis. Syphilis or some valvular disease of the heart is not

infrequently made out in patients who develop this symptom complex. The onset of cerebral pseudobulbar palsy is characterized by its abruptness, very similar to that of an apoplectic stroke, which may or may not be attended with any limitation of consciousness. The bulbar symptoms increase up to a certain degree, and then they remain stationary or begin to improve, until a second attack, or a succession of attacks, greatly intensifies them or terminates the patient's life. The apoplectic attacks are usually accompanied by a hemiplegia of one or of both sides, and this may be transitory or permanent.

The uniform progressiveness of chronic or degenerative bulbar palsy is not a feature of this symptom complex. The clinical condition that follows after the first apoplectic attack remains the same or improves until the second attack intensifies it and perhaps causes death.

The psychical and somatic accompaniments of vascular brain lesion, such as emotional instability, mental enfeeblement, and spasticity of the extremities due to secondary degeneration with its natural concomitants of exaggerated reflexes, are absent in progressive bulbar paralysis and form a part of the symptom complex of cerebral pseudobulbar paralysis. In the latter disease atrophy is never a striking symptom, and when it does occur it is especially to be remarked that it is not true degenerative atrophy, such as occurs in progressive bulbar paralysis, but mere inactivity atrophy. It is a form of atrophy that is not accompanied by the phenomenon of reaction of degeneration. It will be remembered that when atrophy follows disorganization of the central motor neurons of a part, such atrophy is not a true degenerative one, nor does it have the characteristics of the latter. The attitude and gait of the patient are not the same in the two diseases. In true bulbar palsy the head is bent forward, the lower part of the face is immobile, the lower lip hangs, and a handkerchief is held to catch the saliva, while the patient walks without manifest paralysis of the extremities. In pseudobulbar palsy, on the other hand, there is often a symmetrical involvement of the face and observable paralysis of some of the extremities. A symptom that is often very striking in the last-named disease is the spasmodic laughing and crying which these patients often have, and which is in marked contrast to the equanimity of the emotions in chronic bulbar palsy. The speech defect is also different in these two diseases, while dysphagia, a prominent and early symptom of genuine bulbar palsy, may be, and often is, absent in the spurious form.

Taking it all in all, it is probable that the confounding of these two diseases need rarely occur if the history and symptoms of the

patient be carefully inquired into, and especially if opportunity be given to observe the course of the disease for a time.

What has been said concerning the diagnosis of cerebral pseudobulbar palsy applies also to radicular pseudobulbar palsy. In the latter the onset is sudden and apoplectiform, while the clinical symptoms betray evidences of involvement of the pyramidal tracts, such as crossed hemiplegia or alternate hemiplegia, a condition sometimes absurdly called the syndrome of Weber.

Pseudobulbar palsy of neural origin is to be differentiated by its mode of onset, by the progress of the disease, and by certain clinical manifestations. It is unnecessary to speak of the *rationale* of the symptoms, as it is readily seen that a simultaneous lesion of the roots of the ninth, tenth, eleventh, and twelfth nerves immediately or remotely after their extrabulbar origin will produce symptoms similar to those resulting from lesion of the nuclei of these nerves. Such involvement is, as a rule, a part of a general polyneuritis and almost always consecutive to some acute infectious disease. The development of symptoms is much more rapid than in the genuine progressive bulbar palsy, the atrophy of muscles is more severe and attended with more pronounced reaction of degeneration, and frequently there are sensory disturbances in the area of distribution of some of the involved nerves. The symptoms after a time reach a point where no further progression is made, and this in contradistinction to the almost always uninterrupted course of true bulbar palsy. The eventual outcome in the former, although gloomy, is not universally fatal as in the latter. The factors necessary for the exclusion of the dynamic form are given in the description of asthenic bulbar paralysis.

Double facial paralysis can be excluded by the absence of involvement of the tongue, the pharynx, and larynx; by the development and course of the disease, which is generally first of one side of the face and then of the other; by the prompt appearance of reaction of degeneration; by the manifestation of disease in all the musculature supplied by the facial, that is, the actual facial group, the elevators and compressors of the labial commissure and the orbicularis oris group; and by the termination of the disease, which is almost always in at least partial recovery. In pharyngeal and palatal paralysis which sometimes follows the acute diseases, although the nasal intonation, regurgitation through the nose, and dysphagia may be present, the serious and constant manifestation of bulbar palsy, such as universal amyosthenia, loss of prehensile power of the lips, paresis and atrophy of the tongue, etc., are absent.

It is to be differentiated from progressive myopathy of the facio-scapulo-humeral type by the fact that the latter develops in early

childhood; by the "facies myopathique," the widely uncovered, staring eyes, the tapir mouth, and by the distribution of the atrophy, a distribution indicated by the above designation, and by the presence of reaction of degeneration exactly commensurate with the amount of atrophy. Cases of tabes and syringomyelia in which bulbar symptoms supervene are to be distinguished by eliciting the pathognomonic accompaniments of these diseases, among which are to be considered, for the former, the loss of the pupillary light reflex, uniformly contracted pupil, loss of myotatic irritability, particularly of the lower extremities, and absent knee jerk, sensory and sphincter deficiencies, etc., and for the latter the association of a progressive muscular atrophy of certain parts of the body depending upon the seat and extent of the lesion, with profound sensory disturbances, of which disassociation of temperature sense, and analgesia have been considered pathognomonic. Dilatation of the central canal in the medulla and gliosis of it and its continuation, the fourth ventricle, can give rise to symptoms very like those of chronic bulbar paralysis, but the prominence of sensory disturbances should prompt a differential diagnosis. If one of the islets which collectively form the anatomical foundation of multiple sclerosis develops in the ventrolateral area of the oblongata, symptoms simulating those characteristic of bulbar paralysis will develop, or if this be the location of the first anatomical manifestation of the disease, bulbar symptoms will precede the other and diagnostic symptoms of the latter disease, among which are to be considered measured, staccato, syllabic speech, nystagmus, universal exaggeration of tendon jerks, intention tremor, and spasmodic paraplegia. Amyotrophic lateral sclerosis supervening on or preceding chronic bulbar paralysis is to be diagnosed by the presence of symptoms pointing to involvement of the spinal pyramidal projections, such as spasticity, increased myotatic irritability, and the presence of atrophy in the extremities, for the explanation of which disease of the anterior horn cells of the spinal cord must be postulated.

Tumors within the medulla oblongata are so extremely rare, and the symptoms to which they give rise are so different from those of chronic progressive bulbar paralysis, that a consideration apart from the description of these is not warranted. Neoplasms in the basilar region may give rise by pressure on the oblongata to bulbar symptoms, but in addition there are symptoms which bespeak a widespread involvement; such are pain of a neuralgic character in the head and neck, sensory shortcomings, disturbances of equilibration and purposive movements, vertigo, ocular palsies, stiffness of the neck, etc. Frequently the course of the disease, particularly if syphi-

litic, is rapid, and a history of a previous luetic infection may bear evidence contributory to the diagnosis. Slowly growing tumors of the oblongata occurring in childhood sometimes produce symptoms that cannot be differentiated from those of progressive bulbar paralysis.

Pathology and Morbid Anatomy.

The essential pathological change on which the symptoms of this disease are dependent is a progressive degeneration of the nuclei of the motor cranial nerves situated in the lower part of the oblongata. The degenerative changes are most marked in the nuclei of the hypoglossal nerve, and in the spinal accessory; less so, but in many instances very pronounced, in the nucleus ambiguus, the motor glossopharyngeal nucleus, and the nucleus common to the ninth, tenth, and eleventh, called the glossopharyngo-vago-accessorius nucleus. The inferior facial nucleus and the motor trigeminal nucleus are less often involved. The lesions in the oblongata reveal themselves only on microscopical examination. Yet the intra- and extramedullary roots of the cranial nerves most profoundly affected can be seen to be very much smaller, even by the naked eye and with ease by weak magnification. The changes are most profound in the hypoglossal nucleus on both sides, only very exceptionally is the hypoglossal nucleus spared on one side. The cells of this nucleus are the largest in the oblongata, their protoplasmic prolongations the most conspicuous, and their intranuclear network the most distinct; because of these attributes, even though the destructive process may not be more severe than in another nucleus, such as the inferior facial nucleus, the changes are more striking. The structural change consists of shrivelling of all the processes, not alone of the axis-cylinder process of the cell but of the dendritic processes as well. The component structure of the cell undergoes striking changes which are very apparent in sections stained after Nissl's method. In some cells the nucleus is seen to be small and very profoundly stained, while distributed throughout the substance of the cell is a great deal of pigment in the form of clumps or fine granules associated with destruction of the chromophilic substance. In other cells the nucleus will be found to be almost completely absent, and in some cells quite absent, while the normal lamellated appearance of the cell body has entirely disappeared; in its place is a shrunken, irregular, heavily stained mass which structurally bears slight resemblance to the normal cells. With this stain the axis-cylinder prolongations, with their collaterals as well as the dendrites of the cells, are strikingly delineated in normal cells, but in a specimen taken from a case of

bulbar paralysis these structures are stunted and shrivelled and in great part they have disappeared. With the Weigert stain the motor cells of the nuclei show their beginning dissolution by a bright yellowish appearance of all the cell body except the nucleus. This yellow coloration is due to the deposition of granules which are either the result of degeneration and disintegration of the cells, or they are leucocytal basophilic granules. This same stain shows the intracellular fibre network or reticulum to be more distinct than normal but of less delicacy in construction; very often the smaller blood-vessels are proliferated and degenerated, and the perivascular lymph spaces are enlarged. The neuroglia remains unchanged. The white substance of the medulla is intact as a rule. In a number of cases changes in the white substance, particularly in the pyramidal tracts, much less frequently in the fillet, have been reported. Some writers, particularly Gowers and Leyden, contend that degenerative change in the white substance is the rule, and indeed the latter goes so far as to say that destructive changes cannot go on in the cells without lesion of the white substance. This view is not only theoretically untenable but has not received the corroborative evidence of carefully studied cases. Although a comparatively large number of cases are associated with lesion in the pyramidal tracts above and below the medulla oblongata, thus constituting an amyotrophic lateral sclerosis, genuine, chronic progressive bulbar paralysis is a disease of the peripheral motor neurons; and the involvement of these neurons is of their entirety, beginning in cell body and extending throughout the axis-cylinder process or neurite to the terminal end processes in or around the anisotropous actively contractile part of the muscle substance within the sarcolemma of the muscles to which these nerves are distributed. Such limitation of involvement to the peripheral neurons of a limited, functionally associated group of cells entails no accompanying involvement of the central neurons, the motor neurites of which are represented in the oblongata as the pyramidal tracts. If they be involved, and, as has been said, they are sometimes, the disease process in them is a coincident and not a concomitant one. That the lesion in the peripheral neurons of the lower motor cranial nerves is manifest throughout its entire prolongation is shown by examination of the nerve trunks, the motorial end plates, and the muscle substance. They are all the seat of primary degeneration associated with deposition of interstitial sclerotic tissue in the nerve trunks and of fatty tissue in the muscles. All the fibres of the nerve bundle are not in simultaneous stages of decay, no more are all the cells of a nucleus or their protoplasmic and dendritic processes. Side by side with slender, translucent, shrivelled nerve fibres in a trunk are many

that appear quite normal; others, swollen and opalescent, point to beginning degeneration, while others still, looking like sclerotic threads, indicate the completeness of the degeneration process.

The muscular tissue of the tongue, lips, larynx, etc., is found in various stages of atrophic change, corresponding to the degenerated nerve trunks. Here and there will be seen fatty and granular degeneration of muscle fibres surrounded by a great number of normal fibres attended with the deposition of pigment granules, fat, and proliferation of the nuclei of the sarcolemma, while an occasional empty sarcolemma sheath betrays the completeness of the process. Large numbers of connective-tissue cells undergoing granular degeneration, and clumps of reddish pigment lying between the sheaths of the fibres, bespeak the activity of the process, while the interstitial accumulation of adipose tissue indicates the inability to convert this by process of oxidation.

Until a short time ago the explanation of the paralysis of the lips, and of the orbicularis oris, was a very difficult one, and many ingenious theories have been advanced. The one suggested by Lockhart Clarke seemed to be most satisfactory, but it posited anatomical conditions which do not exist, and more extensive research fails to corroborate his views except in a small part. The opinion advanced by this author was that the orbicularis supply was from the small cells immediately above the hypoglossal nucleus, which became diseased coincidently with the hypoglossal nucleus, and this caused the early paralysis of the lips. The investigations of Tooth and Turner on the origin of certain cranial nerves suggest a solution which, if future workers corroborate, gives an explanation of the symptoms in a rational manner because it answers the postulates of anatomy. According to these authors the muscles of the face are divided into the following groups: The oculofacial group, including the frontal, the orbicular, and the superciliary; the group of elevators and conductors of the labial commissure, the zygomatic, buccinator, and risorius, and a group which includes the orbicular of the lips. Those nerve fibres which innervate all these muscles, although all contained in the trunk of the facial nerve, have three separate and distinct origins in the medulla oblongata. The fibres which go to the orbicularis oris arise from cells which are a part of the hypoglossal nucleus, and are carried to the collective facial through the intervention of the posterior longitudinal fasciculus, which is constituted by fibres arising from the oculomotor nucleus.

Thus it will be readily seen that the early and constant involvement of the orbicularis oris is a necessity if the hypoglossal nucleus is involved. This anatomical exposition also makes clear those cases

in which oculomotor symptoms, transitory generally, first of one eye, then of the other, are sometimes the heralding symptoms of this disease. It also explains the unison of action between the orbicularis oris and the free end of the tongue. It is common knowledge that contractions of the one cannot be produced without the indulgence of the other. It must, however, be said that it leaves unexplained those very uncommon cases in which paralysis of the lips occurs with integrity of movements of the tongue. The difficulty of deglutition is dependent upon involvement of the accessory hypoglossal nucleus and the nucleus ambiguus, the motor muscles of the glossopharyngeal, while the laryngeal symptoms in part and the respiratory and cardiac symptoms bespeak involvement of the pneumogastric nucleus.

Duration, Course, and Prognosis.

The duration of the disease is a very variable one. Some cases run a uniformly progressive course and terminate fatally within one or two years. In other cases the course of the disease is characterized by periods of improvement, or at least by remission of some of the distressing symptoms. Such remissions are temporary and have no influence to alter the eventual fatal outcome, although they may add some to the patient's days and comfort. Very rarely, probably never, does the progress of the disease come to a standstill. The course of the disease, as it commonly develops, is essentially chronic, and month after month the gradual increase in the intensity of the symptoms, notwithstanding the most assiduous treatment, is lamentable and discouraging. It is uncommon for the disease to take more than from three to four years to run its course, but it should not be forgotten that a few cases extending over a period of twice that time have been reported.

The prominent immediate causes of death are universal exhaustion, death occurring from heart failure, attacks of syncope, or food pneumonia; foreign substances, principally those taken for alimentation, pass into the larynx and into the respiratory passages, cause strangulation and suffocation, bronchopneumonia, and localized pulmonary gangrene.

The respiratory capacity in every patient with progressive bulbar paralysis is so weakened that the slightest additional encroachment is not tolerated, and the most moderate laryngeal or bronchial congestions, such as would in health be treated indifferently, become of serious moment. Voluntary and forced expectoration are injurious. Patients with this disease learn early the necessity of avoiding causes that produce congestion of the respiratory passages. Paralysis of the

abductors of the vocal cords is a symptom of evil omen, for the danger of suffocation from slight catarrhal swelling of the cord then becomes very great.

Although cases of complete recovery have been reported, such as those of Berger, Keller, and perhaps also of Dana and Hitzig, it seems justifiable from what we know of this disease and its simulators to maintain that such cases were not genuine cases of degenerative bulbar paralysis, but of the form which has since been described under the designation of asthenic bulbar palsy or bulbar paralysis without anatomical foundation. Erb has described cases in which treatment seemed to be effective, but it is now conceded that they belong more accurately to the form of bulbar paralysis just mentioned.

Treatment.

Although this disease leads uniformly to a termination which no therapy has been able to avert, and although oftentimes our most strenuous efforts to delay the fatal outcome are negative, nevertheless in the great majority of the cases not only the patient's comfort can be greatly contributed to, but his existence materially prolonged by assiduous and proper treatment. The most important factors in the treatment are two: keep the patient's nutrition up to the highest possible mark, and give as nearly as possible complete rest to the muscles in which paralysis shows itself. A semisolid and liquid diet should be adopted from the beginning, and this of the most nourishing kind. Milk and its various preparations, eggs, raw or very slightly boiled, the most concentrated meat soups and nourishing gruels should form the principal dietary. The amount of force needed to masticate and swallow meat, and the consequent exhaustion, more than counterbalance any benefit to be derived from it. The proteids, although important tissue-builders, are not so urgently demanded as to warrant giving them in the shape of meat that must be chewed and swallowed. Proteids that admit of being given in liquid form fulfil every requisite. Careful diet lists should be prepared, and the form of food changed with sufficient frequency to prevent the patient from tiring of it. It is a mistake to consider that a larger amount of food than is necessary to keep up the patient's weight is of any considerable benefit. It is found necessary to remove breadstuffs early from the dietary, as they are most liable to enter the glottis and provoke severe spasms of coughing. As soon as swallowing becomes especially difficult, resort must be had to the feeding-tube. The diminished sensibility of the palate and vault of the pharynx which these patients have in the latter half

of the disease contributes to the ease and comfort with which the tube is passed, and it cannot be too strongly emphasized that this method of feeding should not be left until the patient is absolutely incapable of making voluntary deglutitory efforts. This mode of feeding may often be supplemented by limited rectal alimentation. As a rule all forms of alcoholic drinks are harmful in this disease. Their ingestion tends not alone to make the patient more uncomfortable by contributing to palpitation of the heart, flushings, etc., but they have a depressing after-effect which is materially bad. Any beneficial influence they have to stimulate the nutrition is more than replaced by any reliable non-alcoholic malt extract. The same may be said of tea and coffee; cocoa, however, is a nutriment of real value. The patient should be prevented from using his voice with the same scrupulousness as in pneumonia. The early formation of the habit of communicating desires and thoughts graphically can only be advantageous, and is to be commended. Futile efforts to dislodge food that gets between the cheeks and teeth, by the tongue, and all such unnecessary movements, are to be deprecated.

The two therapeutic measures which can be made use of by the physician, with the best prospects of affording some relief, are electricity and strychnine. Various ways of applying the former have been adopted. Personally I believe that any benefit to be derived from this procedure is obtained through its preservative influence on the degenerating muscles and not in any way on the degenerative process in the oblongata itself. Therefore passing the galvanic current from one mastoid process to another, or galvanization of the upper vertebral column, cannot be advocated. The use of the constant current to cause mild contraction in the face, tongue, lips, and pharyngeal muscles, and over the pomum Adami to cause a slight activity of the swallowing musculature, for a few minutes each day is advisable. The patient's comfort and wellbeing are frequently contributed to by a moderate amount of faradization and massage of the extremities. Although the beneficial effects of strychnine are never so apparent in this disease as they are occasionally in its analogue, progressive muscular atrophy of spinal origin, yet it is the most satisfactory vascular and muscular tonic for use in bulbar paralysis. It should not be given hypodermically. The use of iodide of potassium, mercury, and salicylates with the idea of specific and alterative action is a fallacy. Unless a history of comparatively recent syphilis or rheumatism can be obtained, or unless there are other reasons for suspecting these diseases, such drugs are harmful. Nitrate of silver, phosphate of zinc, ergot, etc., have been used extensively, but they cannot be recommended.

Aside from steadying the nutritive balance by restoratives and aids to digestion, and guarding the patient against factors that produce excitement or depression, the treatment is symptomatic. Drooling is but slightly influenced by belladonna and its alkaloid; it is best combated by absolute quiet of the patient.

If paralysis of the vocal cords, or the entrance of foreign substance into the respiratory passages, makes suffocation imminent, one should not hesitate to perform tracheotomy.

Primary Vascular Lesions of the Oblongata.

The blood-vessels of the medulla oblongata are less frequently diseased than the vessels of other parts of the brain. They are, however, sometimes involved and are then accompanied by acute bulbar symptoms, so that it becomes necessary to consider these affections in detail. The pathological processes that cause acute bulbar palsy are acute inflammation, hemorrhage, thrombosis, and embolism, the last two being the most frequent. Whichever of these constitutes the lesion, the symptoms that result are very similar and are to be assigned to the different causes, not so much from the character of the symptoms but from the mode of their onset, from the preceding history of the patient, and from the cause and outcome of the disease. Acute inflammatory bulbar paralysis (poliomyelitis bulbi, acute bulbar myelitis) is the distinct analogue of anterior poliomyelitis, and the little that is known of its etiology corresponds most closely with the causation of the latter affection.

Very few cases in which the diagnosis has been corroborated by autopsy have been recorded. In such, however, as well as in those whose recognition is based on clinical data, the disease seemed to occur in youth and in early adult life and following on or coincident with some acute infectious disease, such as pneumonia, influenza, or diphtheria (Mendel). Alcoholism and rheumatism have been predicated as causative by Leyden, and Reinhold has reported a case of progressive bulbar paralysis in which acute parenchymatous inflammation of the medulla oblongata supervened.

The history of such cases is that the symptoms come on very abruptly, but on close investigation, as in acute poliomyelitis, it is found that prodromal symptoms, such as obscure sensations in the head, dizziness, vomiting, fluttering sensations in the cardiac region, and indefinable feelings of dread and exhaustion, precede the real bulbar symptoms. The onset is sometimes sudden and accompanied by the concomitants of acute infectious processes, such as rigors, shivering, acceleration of the pulse rate, and fever. These are

quickly followed by nasal intonation, regurgitation of liquids through the nose, increasing difficulty of swallowing, exhausting paroxysms of coughing due to the passage of foreign substances into the glottis, paresis of the lingual and buccal musculature, weakness of the extremities, difficulty of breathing, attacks of suffocation, tachycardia, and syncopal attacks.

Occasionally other cranial nerves, such as the sixth, are involved, and in one case the third was affected. In a case reported by Strümpell, there was paralysis of the diaphragm which led up to death.

The course of the disease is a uniformly progressive one and usually terminates in death within the first week. In the very acute form obnubilation of the mental faculties, and hebetude which leads to coma, go hand-in-hand with the development of paralytic symptoms. A subacute or, better, less acute form of this disease, in which the patient survives long enough for manifestations of atrophy to present themselves, occasionally occurs.

Hoppe-Seyler has reported a case of a girl, fourteen years old, who had convulsions when four years old, which persisted eight days. After that time there were paralysis of face and tongue, loss of speech, difficulty of swallowing, and hypersecretion of saliva. The extremities were spared. By the use of the faradic current, and practice at speaking, the patient made some improvement. He thinks the case was one of poliomyelitis bulbi, with lesion in the nuclei of the facial, hypoglossal, and spinal accessory and their nerves. Militating against its peripheral origin are the fact that it began with convulsions, its symmetrical extent, and the absence of reaction of degeneration in the muscles. The stationary course of the disease argued against degenerative bulbar paralysis.

In those cases in which microscopical examination of the nervous system has been made, there have been found foci of inflammation scattered through the oblongata. These are surrounded by spots of capillary hemorrhage and points of softening varying from a minute point up to sufficient size to be recognized by the naked eye. Around these are seen areas which show only infiltration of leucocytes; this with the engorgement of the blood-vessels being the earliest or mildest evidence of the pathological process. If the disease has been very acute and has quickly eventuated fatally, there will be found corresponding destructive changes in the oblongata, and oftentimes in the pons. These may be manifest by a real coagulation necrosis, or a more advanced process still, destruction of the tissues with the formation of cavities. The nerve cells are swollen, robbed of their contour, and their processes are in various stages of destruction. If the disease has persisted for a long time there may be found evi-

dences of connective-tissue overgrowth and changes in the ganglionic cells characteristic of chronic degeneration. In some instances the lesions are in reality those of hemorrhagic encephalitis, one form of which it is.

Hemorrhage into the substance of the oblongata is of exceeding rarity, and except for the bruskness of onset, the rapidity with which the symptoms become most grave, and the celerity with which they lead to a fatal onset, they can in no way be more than suspected during life as differing from thrombus and embolism. It naturally occurs under auspices favoring rupture of vessels in other parts of the brain; these may be summarized in a word—vascular degeneration. A discussion of the specific causes of vascular degeneration need not here be attempted.

Thrombus and embolism are probably responsible for the great majority of sudden or apoplectiform bulbar attacks, the former being of much more frequent occurrence than the latter. The most potent cause of thrombosis is previous luetic infection of moderately recent origin, *e.g.*, ten years. It naturally therefore is seen in early adult life. It occurs also in advanced life and apart from syphilis as the result of the combined effect of diseased blood-vessels, depraved blood, and impaired circulation. Thrombosis is sometimes preceded and predisposed to by some infectious disease which acts to increase and precipitate the coagulative antecedents in the blood. In some rare instances it follows on trauma to the back of the head and neck and diminution of the calibre of the blood-vessels produced by meningitis.

Embolism of the vertebral and basilar arteries may obstruct the blood supply of certain areas of the medulla oblongata as may embolism of the lateral, median, and other arteries of this segment of the central nervous system. As the median artery is distributed with considerable exclusiveness to the nuclear region of the oblongata, it follows that a thrombus or embolus in this artery will be more likely to cause bulbar symptoms than a similar lesion in other vessels.

The causation of embolism of the oblongata does not differ materially from embolism in other parts of the body. The emboli originate in some intracardial inflammation or degeneration. They are of such infrequent occurrence in the branches of the vertebral and basilar arteries going to the oblongata because of the anatomical relationships of the latter. These arteries are given off at nearly right angles from the trunks, and therefore floating masses in the blood are not apt to get out of the direct current and into the small vessels.

The immediate result of thrombosis and embolism is the cessation, or profound perversion of function of the part or parts supplied by

the blood-vessel that is blocked up; the continued and after-effects are the result of foci of softening that occur. These foci may be so small that they can be detected only on microscopical examination or so large as to involve a great part of one lateral area of the oblongata. In specimens in which one focus of considerable size can be seen with the naked eye, the microscope will reveal several small ones about the principal one. As has been pointed out under chronic degeneration of the blood-vessels, simple arterial sclerosis is often a pathological finding to which has been attributed importance from an etiological standpoint, the vascular degeneration preventing proper nutrition, the cell processes and body suffer in consequence. Arterial degeneration of a severe and advanced degree sometimes causes acute bulbar symptoms aside from the presence of a thrombus. It does this either by pressure of its distended inelastic walls on important constituents of the oblongata or by sudden and profound nutritional alteration.

Symptoms.

The premonitory symptoms of acute bulbar paralysis, whether it be due to thrombus or to hemorrhage, are dependent on diseased blood-vessels. Neither of these conditions can occur, unless it be hemorrhage from trauma, without antecedent vascular degeneration. These symptoms are head pressure, particularly in the back of the head, indefinable fears and dread, sensation of stiffness and pain in the back of the neck, dizziness, ringing in the ears, buzzing, thumping sensations in the head, disturbed and unrefreshing slumber, loss of mental and physical energy, etc. Possibly twitching of the muscles of the face or uncontrollable smiling or grinning may precede the attack. For a few days immediately preceding the occurrence of acute symptoms the patient may complain of various undefinable anxiety feelings. On the other hand, premonitory symptoms may be entirely absent, and the attack occurs coincidently with an attack of indigestion or after some acute disease. The first symptom of the attack is generally dizziness of great and increasing severity, usually accompanied with vomiting. The dizziness is so great and is attended by so much mental confusion and inability on the part of the patient to orient himself that he falls to the ground, although there may be, at first, neither paralysis nor disturbance of consciousness. In some instances a similar sense of confusion and dizziness leads very promptly, even before the patient has had opportunity to sit down or call for aid, to rapidly deepening unconsciousness. This unconsciousness may be the forerunner of dissolution which is heralded by failing heart and pulmonary œdema. If the patient re-

covers from the apoplectic attack there will be more or less complete paralysis of the lips, tongue, palate, and laryngeal muscles and the corresponding symptoms depending upon such paralysis. If the lesion be a hemorrhage and of more than very slight extent, there may be paralysis of the extremities and a variable amount of crossed sensory paralysis. The seat of the lesion and its extent will have more to do in determining the symptoms that persist after the stroke than the pathological process on which it is dependent. If the lesion be of the vessel or vessels that supply the ventral portion of the oblongata the symptoms that continue will be those referable to the nuclei involved. As the nuclei there situated are those commonly diseased in Duchenne's paralysis, it follows that the symptom complex will simulate that disease; the symptoms, of course, being much more acute—that is, speech will have a nasal twang, letter and word formation will be defective, there will be great difficulty in swallowing with regurgitation of fluids through the nose; in short, all the symptoms of well-marked bulbar paralysis minus the atrophic accompaniments. Frequently dyspnœa and tachycardia are present even from the beginning.

In some cases the lesion so limits itself, particularly in thrombosis and its consequent softening, that some one nucleus of the group situated near the floor of the fourth ventricle may be spared. This is particularly so in a case which I have had for a long time under observation. The patient presents a typical bulbar palsy, which developed suddenly while convalescing from pneumonia, minus involvement of the hypoglossal nerve. If the hemorrhagic focus or area of thrombosis and thrombotic softening involve the lateral area of the oblongata, there will be—depending upon the extent and severity of the involvement—perversions of sensibility of the opposite side of the body varying all the way from slight paræsthesia up to complete anæsthesia. Indeed, these sensory defects may manifest themselves not alone in one trigeminal area and one side of the body, but it may be on both sides. This was so in a case of acute bulbar paralysis due to hemorrhage into the substance of the oblongata recently reported by Senator. Pain sensibility may likewise be deficient. Ataxia, difficulty in standing and in walking, and impossibility of rapid orientation may likewise be present if the lesion be at all extensive. If these latter symptoms be present, involvement of the restiform bodies or fibres going to them is probable. Difficulty in hearing and unstable equilibration point to involvement of the acoustic root or nucleus. Not uncommonly hemorrhage or thrombosis in the oblongata is associated with similar lesions in the pons. In such cases pontile symptoms will be superadded.

If the patient does not succumb to the stroke, or within a comparatively short period after it, the symptom complex of chronic labio-glosso-laryngeal paralysis may ensue minus its progressiveness. If the lesion has been a limited one, considerable retrogression may follow. The fact that none of these lesions occurs without serious and more or less extensive disease of the blood-vessels indicates that the end is not far removed.

Diagnosis.

The diagnosis of sudden bulbar paralysis is in most cases a possible one, providing the patient recovers from the shock of the stroke, particularly if the symptoms be carefully analyzed and grouped. The differential diagnosis must, however, always remain difficult and very often problematical. In thrombosis the symptoms are often less brusk in their onset and premonitory symptoms, such as vertical cephalalgia, syncopal attacks, loss of mental and bodily vigor, may be more prominent and of longer duration than in hemorrhage. If, however, the process of thrombosis occurs rapidly and the bulbar artery becomes rapidly occluded this will not hold good. The same may be said of embolism. The latter can only be suspected when the locus from which it originates is found. Those cases which survive the shock may develop evidences of secondary degeneration in the central motor neurons, the pyramidal tracts, manifested by contractures, exaggerated reflexes, etc., as in a case reported by Lichtheim, and in one now under my own observation.

Course, Prognosis, and Termination.

As has already been said, the course of the disease, whether it be due to hemorrhage, thrombus, or embolism, is generally to a fatal termination. The larger proportion of cases do not survive the original shock, or if they do they succumb with symptoms of dyspnœa, cyanosis, irregularity of the pulse, or from food pneumonia within the first week. The small number of cases that pass into a more or less stationary, subacute, or chronic stage are principally those due to thrombosis of luetic origin. It may become necessary to differentiate the cases that pass into a subacute stage from pseudobulbar palsy of cerebral origin; but the presence of mental deterioration, the history of two separate attacks of hemiplegia, as well as the occurrence of secondary pyramidal degeneration, the absence of wasting of the tongue, and any evidences of degeneration in the parts supplied by the last four cranial nerves such as "squirming" movements and fibrillary twitchings, speak in favor of the lat-

her condition. It should also be remembered that in these cases the larynx is seldom paralyzed.

If the disease assumes a subacute or chronic course the prognosis as regards life is fairly good. Recovery only very rarely takes place. If after the first few weeks there seems to be a cessation in the activity of the symptoms, and particularly if there be no alarming cardiac or respiratory symptoms, it may be said with considerable safety, and particularly if these conditions have occurred after some acute disease or the moderately recent manifestations of syphilis, that the prognosis is fairly good. Gowers says if at the end of a month there is no sign of improvement, and there is still a considerable degree of paralysis, it is improbable that much improvement will ensue. I have, however, seen a case, in which there could be no doubt as to the diagnosis, begin to improve after the first six months; that is, there was no improvement in the parts supplied by the neurons which bore the brunt of the original lesion, viz.: the nuclei of the ninth, tenth, and eleventh nerves and their prolongations, but the general condition improved so that the patient was a care to no one but himself. The danger of recurrence, and particularly if the vessels be much degenerated, or if the causes of the original attack be operative, should always be borne in mind.

Pathology.

The pathology of these affections has already been referred to under general considerations. In the cases that have come to autopsy, and there are now a considerable number of such, there have been found the customary lesions of hemorrhage or occlusion of the vessel. These processes are fully considered in the article on Cerebral Apoplexy. The seat and extent of the lesion vary in individual cases. In a case reported by Goldscheider of obliteration of the basilar artery accompanied with chronic arachnitis of the fossa of Sylvius, there were recent changes in the upper interolivary fillet, in the motor tegmental area, and in the acoustic nucleus as well as evidences of a previous pyramidal involvement from brain lesion. A striking associate pathological condition in a case described by Reinhold was the presence of innumerable amylaceous bodies all about the area of hemorrhage in the oblongata. A unilateral lesion may cause bilateral symptoms, as has been seen in several instances, notably in cases described by Senator, Leyden, and Gowers. This phenomenon is probably due not alone to the intimate connection existing functionally between the contents of the two sides of the oblongata, but to the destruction of association fibres.

Treatment.

The treatment at the time of the stroke does not differ materially from the treatment of cerebral apoplexy. Absolute quiet, rest, revulsive agents to the back of the head and to the ends of the extremities, and possibly blood-letting are advised. If the physician be so fortunate as to find the patient during the premonitory stage with such symptoms as to make a diagnosis problematical, it then behooves him to take such measures as will operate to lessen the tension on the blood-vessels and steady arterial tone. The careful and judicious administration of nitroglycerin, the bromides and iodides, and very small doses of digitalis combined with absolute rest and a liquid diet will best subserve this purpose.

In every case in which there is a history of syphilis of moderately recent origin (five years) or in which there are any manifestations of such infection, it is absolutely incumbent on the physician to determine how far the administration of iodide of potassium or sodium can be carried without at the same time increasing the coagulability of the blood, an action which it is well known this drug possesses. It is well in all cases, it is believed, to combine this administration with small doses of such substances as are known to regulate blood pressure and with restoratives. If the patient has not had mercurial treatment inunctions should be given up to the point of toleration. In the cases that pass into the subacute stage treatment applicable to the chronic degenerative form of bulbar paralysis is indicated, additional treatment being directed continually to overcome or prevent further progress of the vascular degeneration.

Associated Neuritis of the Bulbar Nerves.

Inflammation of the cranial nerves that arise from the side of the oblongata, called associated neuritis of the bulbar nerves, may occur individually, or may be superadded to an extensive wide-spread polyneuritis. It is an extremely uncommon condition. Cases have recently been reported by Kender and by Modigliano. Disease of the glossopharyngeal nerve never occurs alone, but disease of the ninth, tenth, eleventh, and possibly even the twelfth may occur associated, or individually, from such diseases of the posterior fossa and the membranes covering it as new growths, tuberculous and syphilitic new formations, and from injuries such as stab wounds and infection following them. Degeneration of these nerve trunks occurs, to be sure, in affections of the oblongata in which there is

disease of the nuclei of the nerve, but with such we are not here concerned, as they form a part of those diseases. The variety of associated bulbar neuritis to which we wish particularly to refer is that form arising after some exogenous or endogenous infection, such as diphtheria, leukæmia, beri-beri, etc.

Eisenlohr has reported a case in which the symptoms of bulbar paralysis developed in the course of a leukæmia. In addition to the dysphagia and other bulbar symptoms there were double facial paralysis, anæsthesia in the distribution of the fifth nerve, loss of the sense of smell, etc. Microscopical examination showed multiple hemorrhages in the sheaths of the bulbar nerves as well as extensive lymphoid infiltration into the nerve trunks themselves. Cases in which bulbar symptoms have been superadded to the symptoms of multiple neuritis are sometimes met with. Such a case has recently been reported by Modigliano. In this patient there was intense diffuse atrophy with reaction of degeneration, disturbance of sensibility and severe ataxia, the brunt of the paralysis being in the lower extremities. Bulbar symptoms, disturbance of speech, difficulty of swallowing, incoördination of the anterior facial muscles, unwieldiness of tongue developed later. In this case entire recovery resulted in about a year.

Although a favorable outcome of cases in which symptoms of involvement of the bulbar nerves supervene on an acute infectious disease is very rare, cases terminating in recovery, such as the one just mentioned, are to be found in the literature. The occurrence of such symptoms is of very bad omen.

Diagnosis.

About the only condition from which it is necessary to diagnose associated neuritis of the bulbar nerves is acute bulbar paralysis of inflammatory or apoplectic origin. This will not be a matter of difficulty if it be borne in mind that the former occurs almost invariably during or after some acute infectious disease; that generally the bulbar nerves are involved, not all at the same time, but in succession; that frequently only two or three of the last four cranial nerves are involved, and that some important symptoms constituting the symptom complex of bulbar paralysis are absent. Such, for instance, is a case reported by Guttmann. The symptoms were deviation of the head to the right and drooping of the left shoulder, dysarthric and nasal speech, paralysis of the soft palate, dysphagia, dyspnœa, and tachycardia, but the voice remained unchanged. More or less irregular distribution of the bulbar symptoms, combined with the absence of symptoms of shock, and the early appearance of reaction of degeneration in the

distribution of the involved nerve or nerves, will lead to a suspicion of the disease. The course of the disease will then corroborate or deny this suspicion.

Treatment.

The general management of these cases does not differ materially from that of sudden bulbar paralysis. The same instructions as to rest and feeding apply. The use of a mild current of electricity, either galvanic or faradic, and the administration of strychnine are to be recommended after the acute manifestations have subsided.

Secondary Degenerative Lesions.

Symptoms referable to involvement of certain structural elements in the medulla oblongata occur with several diseases, particularly with (*a*) disseminated sclerosis, (*b*) tabes dorsalis, (*c*) syringomyelia, and (*d*) amyotrophic lateral sclerosis. These symptoms, which do not constitute a clinical entity, are treated of under their several headings, and need only be mentioned here.

If one or more islets of sclerotic tissue, forming a part of multiple sclerosis, develop in the oblongata there may be, depending upon their location and extent, disturbance of mastication and deglutition, trembling of the tongue and lips in addition to the speech disturbance characteristic of the disease. Two symptoms not considered essential to the symptomatology of ordinary degenerative bulbar paralysis have also been found, namely, glycosuria, supposed to be dependent upon the existence of a sclerotic patch near the floor of the fourth ventricle, and polyuria.

The bulbar symptoms of tabes are most frequently referable to the larynx and to a perversion of function of the trigeminal and the glossopharyngeal-vagus nerves. Aside from the occurrence of so-called laryngeal crises, they are manifest principally by disturbance of vocalization and of swallowing, tachycardia, and laryngeal paralysis, particularly paralysis of the posterior cricoarytenoid muscle. Alteration may take place in the ninth, tenth, and eleventh nerves, possibly also in individual nuclei, and when such is the case symptoms referable to disease of these structures will be present singly or collectively; such cases are uncommon, but striking examples have recently been reported by Howard, Charcot, and Chvostek. The symptom denoting involvement of the hypoglossal nucleus is hemiatrophy of the tongue. In some cases of tabes with involvement of the tongue there is found a degeneration of the terminal branches of the hypoglossal nerve in the

tongue, associated with a peculiar interfibrillary fatty degeneration of the musculature of the atrophic side of the tongue.

Bulbar symptoms are more common in syringomyelia than in tabes. They are dependent for their occurrence upon dilatation of the central canal and of the fourth ventricle higher up. The symptoms will depend upon the pathological process, *i.e.*, whether it be a gliomatosis or a myelitic form, and upon the extent of the lesion. In certain cases the bulbar symptom complex which develops is very similar to that of degenerative bulbar paralysis except that the evolution of the symptoms seems to be more precocious and striking. In other cases involvement of only one or two of the lower cranial nerves has been present: in such cases there will be trouble of deglutition, perversion of the sense of taste, disturbance of phonation, hemiatrophy of the tongue, etc.

Bulbar symptoms are common in amyotrophic sclerosis, either as a part of the disease or as terminal symptoms, and they will be considered under the description of that disease.

Tumors of the Oblongata.

Tumors of the oblongata, whether they be glioma, sarcoma, gumma, or tubercle, are of rare occurrence. Glioma is probably the most frequent neoplasm of the oblongata. I have been able to find nine cases of gliomatous formation limited to the medulla oblongata in the literature. The symptoms which they produce depend upon their location in the medulla, the amount of destruction they cause, and somewhat on their size. No detailed description to fit every case can therefore be given. To show the extent to which a tumor may occur in this area, without giving rise to alarming symptoms, the following case may be mentioned:

A young man previously healthy complained eight months before his death of sensation of numbness in the fingers of the left hand which soon extended to the whole upper extremity. Later a similar sensation was felt in the left lower extremity. Aside from this there were no symptoms and he continued at his work. Two months before his death he complained of a feeling of distention in the left side of the head with a dragging, bulging sensation on the same side of the neck. At no time were there vomiting, vertigo, headache, paralysis, convulsions, or symptoms of focal lesion. Strength in the left half of the body had been gradually getting less. Two weeks before his death, in addition to the symptoms just mentioned, all that could be made out was loss of strength on the left side of the body, accompanied by an increase of the deep reflexes, ataxia of the left hand, and universal analgesia, but thermal and tactile sensibility was preserved; tachycardia and a

tendency to filling up of the lungs, which seemed to be periodic, were the terminal symptoms. The patient was up and about the ward until a few days preceding his death, when high temperature (105°), tachycardia, and increasing pulmonary œdema quickly developed and terminated his existence. The growth was of a gliomatous nature which usurped the entire central part of the oblongata, leaving only a thin mantle of nervous substance. It extended from near the pontobulbar junction, caudad as far as the junction of the oblongata with the cervical cord.

In other cases the symptoms have been those of superior and inferior ophthalmoplegia with paralysis of the extremities. It is unnecessary to say that the course of the disease is progressive, its termination fatal, and that all forms of treatment are useless. A possible exception may be syphiloma.

Abscess of the Oblongata.

Rare as are tumors, abscess of the oblongata is rarer still, the only serviceable observations being two cases of Eisenlohr. The infrequency of the disease and the bizarre modes of development make a diagnosis of this condition at the present time impossible.

In the first case reported by Eisenlohr symptoms of left-sided hemiplegia and loss of sensibility in the left forearm developed while the patient was under treatment for left-sided purulent pleuritis. In a very short time the right side became paretic, associated with paralysis of the abdominal muscles. This was quickly followed by death preceded by increasing dyspnœa. In the second case the abscess of the oblongata developed secondarily to, or as a complication of, cerebrospinal meningitis. In this case it was impossible to separate the symptoms due to the complication from those dependent on the original disease. Cultures made in both cases showed a streptococcus infection in the first, and a mixed infection in the second case. Anatomically the pathological process in both was very extensive and destructive.

Progressive Bulbar Paralysis (Infantile and Familiary).

Within the past few years a number of cases of bulbar paralysis occurring in infancy associated with or apart from other defects in the nervous system has been reported. In almost every one of these cases there has existed proof to show that the disease was hereditary and familiary. Such cases have been described by Hofmann, Remak, Fazio, Charcot, Londe, and others. Sufficient is not yet known of the disease to make any considerable description warrant-

able. It has been written of under the above heading, especially by Londe at the suggestion of Charcot. Whether or not future experience will justify such supposition remains to be seen. Progressive bulbar palsy of this type is supposed to be analogous to other familiary and hereditary diseases, a class which experience goes to prove is continually increasing. These cases have heretofore been reported as anomalous types of dystrophy, particularly of the facio-scapulo-humeral type, and as cases going to show that chronic degenerative bulbar paralysis may occur during infancy.

The disease as it presents itself in these patients does not differ so markedly as to justify separate description. It would seem to be characterized, from a symptomatic point of view, by the participation of the superior facial musculature in the paralysis; but, as has been pointed out in discussing chronic bulbar paralysis, this symptom has been occasionally noted in the latter disease, particularly by Remak. This participation of the upper facial musculature is particularly noticed in the cases of familiary bulbar paralysis detected in infancy. As Londe has pointed out, familiary bulbar paralysis in the adult would seem to be unattended with involvement of the upper facial, and has the unusual complication of muscular atrophy especially of the muscles of the neck. In one case reported by Londe there was paralysis of the upper facial with paralysis of the levator palpebræ superioris without external ophthalmoplegia.

As in ordinary chronic degenerative bulbar paralysis, the prognosis is fatal, and the course of the disease is only slightly ameliorated by treatment. It is a well-known fact that acquired diseases run a much more rapid course than inherited, and especially acquired diseases of the nervous system. Under the use of electricity a temporary improvement in some of the symptoms has been reported. The prognosis is generally directly proportioned to the intensity of the disease and the state of nutrition at the onset of the symptoms.

It should be noted in this connection that a form of cerebral glosso-pharyngo-labial paralysis occurs in infancy, and it is very important to separate it from the above-described condition. This is not usually a matter of great difficulty, for these cases are associated with such marked cerebral defects, porencephaly and microgyria, as to prevent the continuance of life for any considerable period, although I am not unmindful that Oppenheim has reported a case that lived till the twenty-first year. Defect of the cortical substance in the vicinity of the central convolutions, especially of their inferior third and of the neighboring portion of the cortex, has in a number of instances produced not alone manifestations of diplegia, but the typical symptoms of bulbar disease. It is well known that a form of cere-

bral diplegia is familiary, and cases of cerebral defect with bulbar symptoms demand examination in reference to this point. It is unnecessary to say that these cases are examples of cerebral pseudobulbar paralysis.

Asthenic Bulbar Paralysis.

(Bulbar Paralysis Without Anatomical Foundation.)

Such is the name which must be given, temporarily at least, to a class of cases in which the symptoms in their entirety resemble very closely chronic degenerative bulbar palsy, and in which after death (the termination to which they eventually lead) examination of the neurons, as well as of the other systems of the body, fails to reveal any striking departures from the normal. The cases of bulbar paralysis which tended towards recovery or periods of long remission described by Erb probably were of this class, but a case described by Wilks is the first in which the absence of anatomical lesion was demonstrated. Although the nervous system in the patient reported by Wilks was carefully investigated, the case was not rated very convincing. It was not until careful examination of the entire central nervous system, as well as of the muscles and peripheral nerves of the parts involved, in cases reported by Oppenheim, Eisenlohr, Hoppe, Shaw, Goldflam, and others, showed the absence of anatomical changes on which the symptoms could be dependent that the symptom complex was established as a disease entity.

Cases analogous to this affection, except that the symptoms predicate a much more extensive involvement of the nervous system, have been reported by Dreschfeld, Goldflam, and others, in which no lesion of the nervous system to account for the symptoms has been found on microscopical examination. In some cases the symptoms are those common to polioencephalomyelitis, while in others they are those of combined superior and inferior polioencephalitis. In Dreschfeld's case the symptoms came on with double ptosis which disappeared after a short time. Six months later a recurrence of the ptosis, diplopia, bulbar symptoms, weakness of the arms, and before long paralysis of all the cranial nerves and upper cervical nerves occurred. Some of the affected muscles underwent atrophy, but electrical irritability remained normal and there were no sensory disturbances. The usual amount of speculation has been indulged in concerning the nature of the lesion in these cases, the preponderance of opinion being that some chronic encephalitic process as yet undetectable by our microscopical technic is at the bottom of them.

As there are less than a half-score cases on record in which post-mortem examinations have been carefully made, it is natural that much concerning the nature and course of the disease is still unknown.

Symptoms.

The symptoms usually develop slowly. The patient may have complained for an indefinite time of easily induced fatigue and a feeling of complete exhaustion after comparatively slight efforts. The development of the symptoms, however, may be rapid and the intensity of the disease reach its height within a few weeks. Frequently the first symptom to give the patient any concern is a ptosis of one or both sides. The ptosis may appear first on one side, then disappear, and the other lid become affected, or it may occur on both sides simultaneously and be associated with paresis of some muscle or muscles supplied by the oculomotor nerve, such as the internal or the superior rectus. Following this, or going before, there occur weakness of the muscles of mastication, paresis of the lower part of the face, defect in articulation and in vocalization, which is associated with paresis of the abductors and adductors of the vocal cords; the voice becomes nasal and continuous efforts at speech are very exhausting and quickly incapacitate the patient for further indulgence. The lips are unwieldy, there may or may not be paresis of the tongue (in fact, the tongue is not infrequently spared), swallowing is difficult or impossible, fluids regurgitate, the soft palate is lax and responds very sluggishly to mechanical irritation. Weakness with a feeling of exhaustion in the trunk and extremities, true amyosthenia of all the motor parts of the body, develops symmetrically, simultaneously with or after the bulbar symptoms. In exceptional cases the weakness manifests itself first in the arms, extends to the legs, and eventually shows itself in the cranial nerves. As the disease progresses, and this it may do with considerable rapidity, respiratory and cardiac symptoms may become very distressing and foreshadow dissolution.

In contrast to true bulbar palsy, the muscles preserve their normal volume, or at least there is no degenerative atrophy; electrical irritability is preserved, but frequently quickly exhausted after brief excitation, and irritability does not then recur until after considerable rest. There are no fibrillary twitchings of the muscles of the face and extremities and the deep reflexes are present, but, like the electrical irritability of the muscles, their excitability is quickly exhausted and recovered only after rest. There are no disturbances of sensibility, either objective or subjective; the special senses are unaffected,

although I have noticed in a case which has been for a long time under continual observation that these as well as the other parts of the body are fatigued readily. Digestion is usually somewhat impaired and normal intestinal action seems to be handicapped by lack of sufficient muscular tone. There is no drooling, and the sphincters are intact; the psychical faculties are not impaired. Although the course of the disease may be a somewhat continuous and rapid one, it is frequently characterized by periods of remission, more rarely of intermission, during which time there is an entire cessation of active symptoms, but the patient probably never regains complete health and strength. This period may last for an indefinite time; indeed it is said by some writers that the disease may terminate in recovery. This latter would seem possible in those cases occurring in youths who, before their illness, were strong and healthy. The shortest duration of the disease is six months; the longest, aside from those cases in which the symptoms are so very slight as not to be considered, six years.

Nothing is known of the causation of the disease. Of the cases reported the majority have been under the age of thirty. It has been observed in a case of profound chlorosis. The possibility that it is dependent upon a chronic intoxication of endogenous or possibly of exogenous origin has been suggested, but the course of the disease does not lend itself to this view. Such is evidently the idea of Jolly, who has described cases which apparently belong in this category under the cumbersome but expressive title of myasthenia gravis pseudoparalytica. In Jolly's two cases the bulbar symptoms, although present, did not predominate over the universal myasthenia; but essentially his cases coincide with those just described, for, as has already been said, in this disease it would seem that in some cases the eye muscles, in others the muscles supplied by nerves arising from the oblongata, while in others the muscles of the trunk and extremities, bear the brunt of the affection.

Diagnosis.

It is necessary to differentiate this symptom complex from the apoplectic form of bulbar palsy, from chronic degenerative bulbar paralysis (Duchenne's type), and from cerebral pseudobulbar palsy. Although it is a comparatively easy matter to make the diagnosis theoretically between organic and non-organic bulbar paralysis, in reality it is very difficult, and the diagnosis of the latter can only be strongly suspected during life, at least it cannot be made with absolute positiveness. If it be borne in mind that in the latter disease there are absence of sensory symptoms, particularly absence of disturbance

of muscular sense, absence of true degenerative atrophy and true reaction of degeneration, absence of fibrillary twitchings and the involvement of the third nerve; that oftentimes the upper and lower facial and the motor branch of the fifth nerve are involved, that the twelfth nerve is frequently spared, and that there is a tendency to cessation and intermittency of the symptoms, the diagnosis of asthenic bulbar paralysis will at least be suggested. The course of the disease must then be studied, to substantiate it.

The differential diagnosis from cerebral pseudobulbar paralysis must always remain a difficult one, as in both there is absence of atrophy in the paretic muscles, and in both the symptom complex may be only atypically that of bulbar paralysis. Cases in which there is a history of one or more attacks of hemiplegia, it matters not how suggestive other symptoms may be, cannot be admitted into this category. It is for this reason that a recent case of Senator's cannot be considered as belonging to this group. The differential diagnosis from associated neuritis of the bulbar nerves will rarely have to be made. The possibility of hysteria must sometimes be considered.

Treatment.

Complete and absolute rest to all parts of the body, the eyes, the tongue, the throat, and the extremities, is the most important factor. Restoratives and the careful and judicious use of measures to stimulate bodily nutrition, at the same time taking every precaution to prevent unnecessary expenditure of energy and bodily waste, will be attended with the best results. Artificial feeding by means of the stomach tube should not be resorted to, as the movements of regurgitation produced by the passage of the tube are more exhausting to the patient than is the act of swallowing artificially masticated and liquid food. Oppenheim warns against the use of electricity for the purpose of causing muscular contraction, but recommends central galvanization. The usefulness of the latter has been corroborated by Goldflam, who reports the recovery of four cases. Jolly mentions the possibility that veratrine used with extreme caution may be of some efficacy.

If it be borne in mind that in this disease all the voluntary muscles, and especially the oblongata musculature are in such a state that slight stimulation soon exhausts them, it will not be necessary to warn against the incautious use of strychnine, galvanism, and massage, the three most available muscular tonics. All of these may be employed if intelligence directs their use. Strychnine should be given in extremely small doses, its effects being carefully watched, and the moment it produces any feeling comparable to fatigue or exhaustion after its

physiological effects have worn off the dose should be materially diminished. Massage and galvanic electricity may, I believe, be used to advantage if sufficient care and attention be given to their application, and if they be given in sufficiently small dosage.

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tendency to filling up of the lungs, which seemed to be periodic, were the terminal symptoms. The patient was up and about the ward until a few days preceding his death, when high temperature (105°), tachycardia, and increasing pulmonary œdema quickly developed and terminated his existence. The growth was of a gliomatous nature which usurped the entire central part of the oblongata, leaving only a thin mantle of nervous substance. It extended from near the pontobulbar junction, caudad as far as the junction of the oblongata with the cervical cord.

In other cases the symptoms have been those of superior and inferior ophthalmoplegia with paralysis of the extremities. It is unnecessary to say that the course of the disease is progressive, its termination fatal, and that all forms of treatment are useless. A possible exception may be syphiloma.

Abscess of the Oblongata.

Rare as are tumors, abscess of the oblongata is rarer still, the only serviceable observations being two cases of Eisenlohr. The infrequency of the disease and the bizarre modes of development make a diagnosis of this condition at the present time impossible.

In the first case reported by Eisenlohr symptoms of left-sided hemiplegia and loss of sensibility in the left forearm developed while the patient was under treatment for left-sided purulent pleuritis. In a very short time the right side became paretic, associated with paralysis of the abdominal muscles. This was quickly followed by death preceded by increasing dyspnœa. In the second case the abscess of the oblongata developed secondarily to, or as a complication of, cerebrospinal meningitis. In this case it was impossible to separate the symptoms due to the complication from those dependent on the original disease. Cultures made in both cases showed a streptococcus infection in the first, and a mixed infection in the second case. Anatomically the pathological process in both was very extensive and destructive.

Progressive Bulbar Paralysis (Infantile and Familiary).

Within the past few years a number of cases of bulbar paralysis occurring in infancy associated with or apart from other defects in the nervous system has been reported. In almost every one of these cases there has existed proof to show that the disease was hereditary and familiary. Such cases have been described by Hofmann, Remak, Fazio, Charcot, Londe, and others. Sufficient is not yet known of the disease to make any considerable description warrant-

able. It has been written of under the above heading, especially by Londe at the suggestion of Charcot. Whether or not future experience will justify such supposition remains to be seen. Progressive bulbar palsy of this type is supposed to be analogous to other familiary and hereditary diseases, a class which experience goes to prove is continually increasing. These cases have heretofore been reported as anomalous types of dystrophy, particularly of the facio-scapulo-humeral type, and as cases going to show that chronic degenerative bulbar paralysis may occur during infancy.

The disease as it presents itself in these patients does not differ so markedly as to justify separate description. It would seem to be characterized, from a symptomatic point of view, by the participation of the superior facial musculature in the paralysis; but, as has been pointed out in discussing chronic bulbar paralysis, this symptom has been occasionally noted in the latter disease, particularly by Remak. This participation of the upper facial musculature is particularly noticed in the cases of familiary bulbar paralysis detected in infancy. As Londe has pointed out, familiary bulbar paralysis in the adult would seem to be unattended with involvement of the upper facial, and has the unusual complication of muscular atrophy especially of the muscles of the neck. In one case reported by Londe there was paralysis of the upper facial with paralysis of the levator palpebræ superioris without external ophthalmoplegia.

As in ordinary chronic degenerative bulbar paralysis, the prognosis is fatal, and the course of the disease is only slightly ameliorated by treatment. It is a well-known fact that acquired diseases run a much more rapid course than inherited, and especially acquired diseases of the nervous system. Under the use of electricity a temporary improvement in some of the symptoms has been reported. The prognosis is generally directly proportioned to the intensity of the disease and the state of nutrition at the onset of the symptoms.

It should be noted in this connection that a form of cerebral glosso-pharyngo-labial paralysis occurs in infancy, and it is very important to separate it from the above-described condition. This is not usually a matter of great difficulty, for these cases are associated with such marked cerebral defects, porencephaly and microgyria, as to prevent the continuance of life for any considerable period, although I am not unmindful that Oppenheim has reported a case that lived till the twenty-first year. Defect of the cortical substance in the vicinity of the central convolutions, especially of their inferior third and of the neighboring portion of the cortex, has in a number of instances produced not alone manifestations of diplegia, but the typical symptoms of bulbar disease. It is well known that a form of cere-

INTRACRANIAL HEMORRHAGE, EMBOLISM, THROMBOSIS (APOPLEXY AND HEMIPLEGIA).

FEW terms in medicine have caused more confusion than that of *apoplexy*. As originally defined and used, it was the name given simply to a certain complex of symptoms, and it brought to the mind's eye the clinical picture of a man who suddenly falls to the ground, with loss of consciousness, labored breathing, a tense, slow pulse, flushed face, and some degree of paralysis upon one side of the body. In course of time the pathological anatomists found that this condition was often associated with hemorrhage into the brain, and hence the term *apoplexy* came to be used as identical with the pathological condition, cerebral hemorrhage. Later it was seen that the brain lesion was sometimes an acute softening, rather than a hemorrhage, and at other times there was found perhaps only an intense hyperæmia or an œdema, or perhaps no lesion at all; hence the terms hemorrhagic apoplexy, serous apoplexy, nervous, congestive, and embolic apoplexy were employed. The use of the word was then still further extended and made to apply to the pathological condition alone, so that authors began to speak of an "apoplexy" of the lungs or of the kidney or of the liver. One can resolve this confusion best by simply remembering that apoplexy properly speaking indicates a group of symptoms alone and not any special pathological state. But these clinical symptoms of apoplexy are caused by the sudden occurrence of lesions in the brain, and, practically, these lesions are always a hemorrhage, an embolism, or a thrombosis. Hence I shall use the term only to indicate that sudden loss of consciousness, with usually more or less paralysis, that is produced by cerebral hemorrhage or by acute cerebral softening, due to an embolism or thrombosis, or in rare instances by a functional brain disturbance.

The apoplexies are almost invariably followed either by death in a few days or by partial recovery, with the development of paralysis upon one side of the body, or *hemiplegia*, and sometimes by a striking set of symptoms, which are included under the term *aphasia*. In studying this subject we have, therefore, upon the one side, to

consider the clinical symptoms of apoplexy in its various forms with the consequent hemiplegia, aphasia, and numerous minor conditions; on the other hand, we have to consider the pathological conditions underlying these symptoms.

In the record of vital statistics it has been the custom for many years to divide apoplectic attacks and their results into two groups—apoplexy and paralysis; apoplexy being put down as the cause of death when death occurred directly as the result of the shock; paralysis being put down as the cause of death when the patient died from some local or half-sided paralysis later.* This system of nosology is grossly inaccurate, but was perhaps the best that could be made at the time it was introduced.

As apoplexy may be due to either a cerebral hemorrhage, an embolism, or thrombosis, it is convenient to speak of hemorrhagic, embolic, and thrombotic apoplexy. This classification was adopted by Lidell, who added, as have other writers, such terms as serous apoplexy and nervous apoplexy. Serous apoplexy is a condition with which I have never personally met, and I have seen no reason for believing in its existence as a distinct nosological entity. It is probably a manifestation of uræmia. There is a form of apoplexy produced by hysteria in which the patient suddenly falls to the ground, apparently unconscious, and becomes paralyzed upon one side. These cases sometimes simulate true apoplexy, and the term *hysterical apoplexy* is a justifiable one.

History.

Apoplexy has been observed since the time of Hippocrates, and in those early days it was, as now, a striking and serious visitation. Hippocrates called apoplectics "those who in health are taken with pains in the head, fall down, become suddenly deprived of speech, and have stertorous respiration. They die," he says, "in seven days or less, if fever sets in." Galen gave a similar description, except that he did not include headache among the symptoms of the attack. Apoplexy was described by Aretæus and referred to by Celsus and Paulus Ægineta. Wepfler, in 1675, showed that in apoplexy there was a hemorrhage in the brain, and Morgagni published many reports confirming this observation. The pathological anatomists of the eighteenth century devoted a great deal of study to this subject, and did much to elucidate it. Up to the year 1820, according to

* Themison distinguished between apoplexy and paralysis: in apoplexy there was a loss of consciousness and paralysis, while paralysis was merely a sudden onset of hemiplegia, without any loss of consciousness.

Frank, quoted by Nothnagel, the apoplectic stroke had been described by more than two hundred and fifty writers. Abercrombie, in the last century, Rokitansky and Virchow in the present century, were among the first to show that apoplexies were dependent primarily upon diseases of the cerebral blood-vessels. This subject was worked out later with great fulness by Charcot and Bouchard. The symptoms of apoplexy were fully studied in the last century also by Boerhaave and his school in Holland, by Abercrombie and Todd in England, and by Rochoux and Andral in France. Among the American writers, much credit is to be given to Dr. John A. Lidell, whose treatise on apoplexy is a most learned and valuable one.

Hemorrhagic Apoplexy.

Etiology.

Age.—Apoplexy from cerebral hemorrhage occurs with relative frequency in the first year of life. After this, the number of cases drops very rapidly, and very few occur up to the fifteenth year. From the fifteenth year the number of cases of apoplexy of all forms—that is, hemorrhagic, thrombotic, and embolic—steadily increases. In a table of deaths from apoplexy, including hemorrhage, thrombosis, and embolism, in the city of New York, from 1866 to 1893 inclusive, there were altogether 16,892. I have made a table based upon these records furnished me by the New York City Board of Health. They show that the deaths occur with increasing frequency up to the decade between 45 and 55, during which time 23 per cent., or nearly one-fourth of the cases, occur. In the next decade, 55 to 65, 20 per cent. of all deaths due to apoplexy occur. Taking into consideration the smaller population at this age, the number of cases is practically about the same. After the age of 65, however, the number of deaths from apoplexy slightly diminishes, being 12 per cent. of the total, and in the last decade, 75 to 85, only 2 per cent. of all apoplexies occur.

	First year.	First five years.	6-15.	16-25.	26-35.	36-45.	46-55.	56-65.	66-75.	76-85.
Absolute number in each decade.	353	495	165	1,181	1,915	3,333	3,922	3,506	2,013	362
Percentage on total.....	2.08	2.8	.09	7.	11.	19.	23.	20.	12.	2.

Occupation.—Apoplexy from cerebral hemorrhage occurs in cities rather more frequently among mechanics, artisans, and salesmen and

those who lead an indoor life, than it does in those who lead an outdoor life, even though that be accompanied by a good deal of exposure and exertion. Occupation, however, influences apoplexy more on account of its influencing habits than because the occupation itself has any special tendency. Thus those occupations which lead more to indulgence in alcohol and more to exposure to venereal disease, will have a larger percentage of apoplexies.

Season and Climate.—Cerebral hemorrhage is slightly more frequent in winter than in summer, and in temperate climates than in the torrid zone.

Heredity.—There is, undoubtedly, a certain amount of hereditary influence in the production of apoplexy, but this is rather an influence towards the production of arterial disease than a direct inheritance of apoplexy.

Alcohol.—The use of alcohol is one of the strongest of the predisposing causes to apoplexy, and indeed, an excessive indulgence may be the actual exciting cause of it. The use of tobacco has probably little influence in producing the disease.

Syphilis is a very important factor indeed in the production of apoplexy. It may be considered perhaps an exciting rather than a predisposing cause, since it not only causes diseases of the vessels, but it actually leads to acute conditions which cause them to rupture. Syphilis, however, is a more important factor in causing thrombosis and softening than it is of hemorrhage.

Bright's disease, gout, the infectious fevers, such as scarlet fever, diphtheria, small-pox, typhoid fever, all produce at times disease of the cerebral blood-vessels, and predispose in this way to apoplexy. Purpura hæmorrhagica and scurvy are also causes.

Certain congenital conditions of the blood-vessels, probably, in some cases, lead to apoplexy. Such conditions are undue narrowing or widening of the carotid arteries or of the aorta.

Disease of the heart is sometimes indirectly the cause of apoplexy, by reason of the disturbance in blood pressure, and partly by producing cerebral embolism, and thus later hemorrhages.

Cerebral hemorrhages have seemed to me, from a study of my hospital statistics, to occur rather oftener in the morning or in the evening hours, and more rarely in the middle of the day. It has been stated that cerebral hemorrhages occur during sleep, and it is not infrequent for patients to go to bed apparently well, and wake up in the morning to find one side of the body paralyzed, but these are usually cases of thrombosis.

The *exciting causes* of cerebral hemorrhage and apoplectic strokes

are a sudden exertion, such as lifting heavy weights, the indulgence in violent passion, great mental strain, overloading the stomach with food, and the ingestion of large amounts of water, and especially excessive indulgence in alcohol. Falls and blows on the head may also lead to rupture of cerebral blood-vessels.

In many instances, attacks of cerebral hemorrhage seem to come on without any cause. It has been shown that in many cases, before the onset of cerebral hemorrhages, there has been for some time a gradual rise in the general blood pressure which has thrown an undue weight upon the work of the cerebral blood-vessels.

Symptoms.

The immediate symptoms of an attack of apoplexy vary somewhat in accordance with the extent and location of the hemorrhage. In an ordinary case of cerebral hemorrhage the phenomena are somewhat as follows: The patient is often at the time of the occurrence feeling quite well; in some instances, however, his attack is preceded by sensations of fulness in the head, perhaps slight nose-bleed, sometimes by a somewhat persistent headache. He may have had insomnia for a few nights or disturbed sleep and bad dreams, and may have suffered from feelings of numbness and prickling in one side of the body. These premonitory symptoms, however, are much more frequently associated with thrombosis and softening than they are with intracranial hemorrhage. With or without such premonitory symptoms, the patient suddenly experiences a feeling of fulness and dizziness in the head, and then falls down unconscious. He is lifted into the bed, and then found to be suffering from a paralysis of one side of the body—this paralysis involving the arm and leg, and to some extent the face. The eyes are partly closed; the face is flushed; the heart beats slowly (50 to 60 per minute); the pulse feels hard and full; the carotids can be distinctly felt, and perhaps seen, throbbing in the neck; the respiration is slow and stertorous. On the paralyzed side the cheek is blown out with each expiration. On lifting the paralyzed arm it falls helpless to the side, and if the leg is drawn up it also drops down limply upon the bed again. On pricking or pinching the patient, some manifestation of consciousness is sometimes shown; the leg may be jerked up slowly, but the arm often remains helpless, and the patient simply carries the sound arm over and tries to push away the hand of the person who is producing the painful irritation. The pupils of the eyes are generally contracted and rather immobile, and one pupil may be, and often is, a little larger than the other, the pupil being more contracted on the sound

side than on that which is paralyzed. On taking the temperature in the rectum it may be found that it has fallen two or three degrees, being 96° or 97° F., though this is not often the case; in four or five hours it will be found about normal, or a degree higher than normal, and it continues thus for a day or more after the onset of the disease, being one-half to one degree higher on the paralyzed than on the sound side. The patient may continue in this comatose or semi-comatose condition perhaps with relaxed sphincters for several hours or days; he gradually, however, becomes more conscious, recognizes friends, answers simple questions, begins to swallow food, and shows a general improvement in symptoms. At the end of twenty-four hours he may be able to move the leg a little, and perhaps the arm. The breathing is less stertorous, and the pulse has become more rapid and regular and softer. Within three days, if the patient is going to recover, he will have regained to some extent his consciousness, and with it some slight mobility on the affected side. With returning consciousness it may be found that there is loss of sensation upon the paralyzed side, but this is rarely complete unless the hemorrhage is a most severe one. By the third day the apoplectic patient usually begins to show signs pointing either to a recovery from the attack or to a fatal issue.

In the former case there is a gradual subsidence of the temperature, both in the rectum and on the paralyzed side; in other words, the general and unilateral disturbance of temperature becomes less, consciousness gradually returns, and the patient is able to speak, to describe his condition, and to understand the conversation addressed to him. The paralysis improves slightly, although it still remains very considerable, and by this time there may be a slight increase in the deep reflexes of the affected side. The pulse becomes normal, the pupils even, the patient swallows and digests his food. There is a steady improvement in all symptoms, until at the end of from four to six weeks he has reached a very considerable degree of recovery. At this period he enters upon what might be called the chronic stage, which I will describe later. In cases in which the lesion is in the left hemisphere the patient has a disturbance in the faculty of language, known as aphasia; he is unable to express his ideas, although he understands what is said to him; or, on the other hand, he may be able to talk a little, but he skips various words and puts them together wrongly, and is in a condition which is known as paraphasia; or, finally, he may be able to talk intelligently, and express his wants by writing, but he is unable to understand what is said to him, is unable to read or unable to recognize the use of things. This condition, which is known as sen-

sory aphasia, is often associated with some degree of anæsthesia on the paralyzed side, and with some hemianopsia.

If the patient at the end of the third or fourth day does not begin to improve, new symptoms usually develop. His temperature begins to rise again until it may reach in a few days 102° or 103° F., being, as a rule, still a little higher on the paralyzed side. He becomes unconscious again, or lies in a state of muttering delirium, being restless, throwing his sound limbs about, pressing his hand to his head, apparently suffering from pain. He will often in this condition pass his fæces involuntarily, the mouth becomes foul with saliva, and deglutition is often difficult. The restlessness and delirium may pass into a condition of coma, and in this state the patient dies within four or five days or a week. Sometimes the patient lingers on a week to ten days, but rarely over this latter period of time. In many instances a pneumonia develops which seriously complicates the condition and hastens the end.

The phenomena of an attack of apoplexy vary, as I have already stated, in accordance with the extent and location of the hemorrhage; but when the hemorrhage is extensive it usually breaks into the ventricles, and then is almost uniformly fatal, and the symptoms, when this occurs, are those which I have just described as being characteristic of an ordinary fatal attack of hemorrhagic apoplexy.

Different Clinical Types.

Hemiplegia of Arm Type.—In some cases it is found that the arm is particularly singled out, so that it becomes very rigid, very much contractured, and practically a useless member. The leg and face are but slightly involved. If the lesion is upon the left side, a very complete motor aphasia is usually associated with this.

Hemiplegia of Leg Type.—In some instances the apoplectic attack is followed by a hemiplegia in which the leg is mainly involved, instead of the arm. In this case there is usually associated with it cutaneous anæsthesia, and at times a hemianopsia. Here the lesion has involved the posterior part of the posterior portion of the internal capsule. In other cases the patient, after suffering from an apoplectic attack, recovers almost entirely the use of the limbs on the affected side, so far as motor power is concerned. He finds, however, that the movements of the affected side are clumsy and incoordinate, and that there is a considerable amount of anæsthesia. He has, in other words, a hemiataxia and hemianæsthesia, with little loss of power. There may be also a hemianopsia. If the lesion is upon the left side of the brain in these cases, sensory aphasia is present, showing itself in the form of word blindness or soul blindness.

The lesion here has involved the most posterior part of the internal capsule, taking in the optic fibres and the sensory tract.

Other and rarer types of hemiplegia are the following:

Hemiplegia with Hemichorea and Athetosis.—Here the lesion

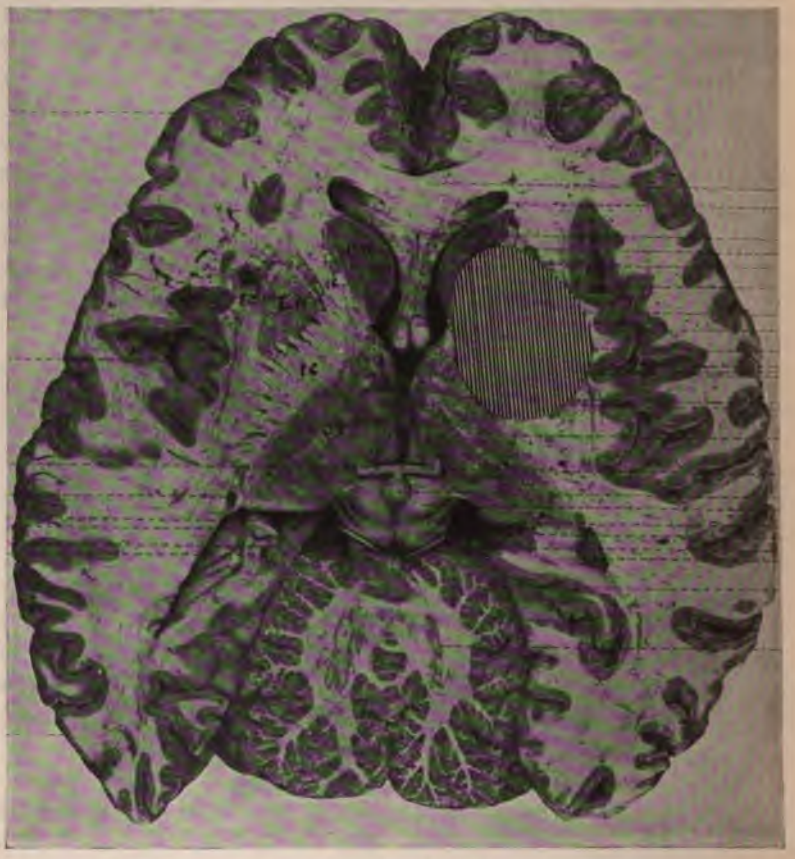


FIG. 21.—Showing the Part most often Affected in Cerebral Hemorrhage. *2m*, *2s*, Internal capsule; 1, knee of capsule carrying fibres from operculum of Rolando; *2m*, motor, *2s*, sensory part of capsule; *T*, bundle of Türck from temporal lobes; *O*, retrolenticular bundle containing optic fibres.

involves the putamem and optic thalamus and invades, to some extent, the middle of the posterior part of the internal capsule.

Hemiplegia with Hemianopsia and Facial Paralysis.—In these cases the lesion is narrow and long, involving the neighborhood of the knee of the internal capsule and extending back through the lenticular nucleus so that it reaches the posterior part of the posterior portion of the internal capsule.

Hemiplegia with Hemianæsthesia and Logoplegia.—Here the patient has loss of power upon one side, with some anæsthesia, and a paralysis of the muscles of articulation, so that he is unable to use the throat, lips, and palate for purposes of speech. This is a rare type, and is due to a total obstruction, probably, of the fibre from the operculum of Rolando running in the knee of the internal capsule and also involving the whole extent of the motor and sensory tracts behind it. (See Fig. 21.)

Ingravescent or Progressive Apoplexy.—This is a form characterized in its onset by sudden headache, vertigo, and sometimes vomiting, but without loss of consciousness. Complete hemiplegia, with some hemianæsthesia, rapidly sets in. The patient in the course of a day or two becomes somnolent, stupid, and finally comatose. Death occurs at the end of from one to five days, with severe respiratory disturbances and rise of temperature. The special characteristics of this type are the onset of a severe hemiplegia without loss of consciousness, its progressive character and fatal termination. It is due to a hemorrhage, which involves primarily the white matter just behind the outer segment of the lenticular nucleus. It cleaves its way forwards into the external capsule. It also extends backwards into the internal capsule at its posterior part and then breaks into the lateral ventricle. The vessel affected is one of the posterior branches of the external lenticular artery.

Various other combinations of paralysis with sensory disturbances and aphasia occur; but, after all, the vast majority of cases present the common type which I have described, associated with some form of aphasia if the lesion is upon the left side.

Symptoms of Hemorrhagic Apoplexy due to Lesions of the Meningeal Arteries, the Cerebellum, and the Pons.

Aside from the apoplexies due to rupture of the central arteries and involvement of the basal ganglia just described, there are a minor number in which the meningeal arteries, the cerebellar, or some branch of the basilar are affected; hence we have meningeal, cerebellar, and pons hemorrhages.

Meningeal Hemorrhage.—Hemorrhages from the vessels of the dura mater are usually due to a rupture of the middle meningeal artery or vein or some of its branches, and this is especially true in such hemorrhages as are the result of injuries to the head. The causes are injuries to the head, including obstetrical injuries, alcoholism, and insanity.

In dural hemorrhages the *result of head injuries,* the clot is sometimes intradural, lying in the arachnoid space, and sometimes epi-

dural, lying between the bone and the dural membrane. The extradural hemorrhages are perhaps a little more common. In over one-half of these there is an interval of consciousness lasting from a few hours to two months, but usually only a few hours, between the accident and the time when distinctive cerebral symptoms develop. Then the patient gradually becomes dull, somnolent, and finally comatose. In about one-half the cases this interval of consciousness between the accident and the development of hemiplegia is present. Along with the gradual or rather sudden loss of consciousness there develops a hemiplegia upon the side opposite the clot. This is usually not complete, though it may become so. Anæsthesia is rarely present. The reflexes are generally somewhat exaggerated, and there may be considerable rigidity. Spasmodic movements of some kind occur in nearly half the extradural cases and in more than half of the intradural. These spasmodic movements may involve the whole of the affected side, or may simply affect the eyes and the facial muscles. They consist of irregular twitchings. The pupils are usually somewhat contracted, more so upon the paralyzed side. When there is a dilated pupil on the side of the lesion and a small pupil on the opposite side, it is known as the *Hutchinson pupil*, and means a severe brain compression involving the third nerve at the base. The eyes are generally both turned towards the affected side and away from the lesion. The pulse is slow and full; the respiration is rarely stertorous, though it may sometimes be so, and Cheyne-Stokes respiration may be present. In these cases the clot is very large and the compression great. Aphasia may be present if the clot is upon the left side. The temperature may be raised one or two degrees, or it may be normal. The progress of the disease is usually steadily fatal unless surgical interference is undertaken. The coma deepens, the respiration becomes stertorous and then embarrassed, the pulse gets rapid and weak, and the patient dies. With surgical interference (since 1886), between two-thirds and three-fourths of the cases are saved (Scudder and Lund).

Dural hemorrhages, *occurring idiopathically*, are due sometimes to the rupture of a meningeal artery, and sometimes to rupture of the veins of the pia mater. This idiopathic hemorrhage is rare in ordinary practice, but is not specially so in insane asylums or in large city hospitals. This is because the two great causes of this type of hemorrhage are insanity and alcoholism. General paresis is the form of insanity with which it is oftenest associated. In the case of alcoholics, it is probable that injuries from blows are an exciting factor in the production of the hemorrhage, these occurring while the

patient is in a state of intoxication. The symptoms of idiopathic hemorrhage are extremely variable, owing to the complicating influences of the insanity and alcohol. The patient after suffering from headaches or vertigo becomes suddenly comatose and shows marked evidences of hemiplegia and even of anæsthesia. Rigidity of the paralyzed side is often present, and sometimes spasmodic movements are observed. On the other hand, at times the paralysis can hardly be observed, and the patient is in a semicomatose state, has a muttering delirium and presents the general aspect of a person suffering from the œdema or "wet-brain" of alcoholics. In dural hemorrhages occurring in paresis, the patient usually without warning becomes unconscious, and he often has some convulsive symptoms and a hemiplegia develops. In these cases there is often a rapid improvement, and the patient gets partly well, usually experiencing other attacks later.

Pial hemorrhages occur very rarely, and the most frequent causes are trauma associated perhaps with syphilis and alcoholism. In many instances very slight localizing symptoms occur, and no absolute diagnosis can be made. If the hemorrhage, however, is in the motor area of the cortex, local spasmodic movements and some hemiplegia are observed. The most characteristic symptoms are the sudden incomplete hemiplegia, involving, perhaps, mainly an arm or a leg, associated with local spasmodic movements, resembling Jacksonian epilepsy.

Pons Hemorrhages.—These are accompanied with initial loss of consciousness and sometimes with spasmodic, jerking movements of the limbs, more particularly of the legs. Some rigidity on both sides of the body may be present. The speech, articulation, and swallowing may be affected. The pupils are often contracted almost to a pinpoint, and the respiration is slow. The temperature almost always rises, and may reach as high as 103° or 104° F. There may be some disturbance in sensation and some hemiplegia. These hemorrhages are usually fatal.

Cerebellar Hemorrhage.—Hemorrhage into the cerebellum occurs in one or two per cent. of all fatal cases. Its recognition is very difficult. There is sometimes a short preliminary period of severe headache. Loss of consciousness occurs almost invariably. The patient falls into a state of profound coma, with stertorous respiration. Vomiting sometimes occurs. There may be some hemiplegia, and if so, this is on the side of the lesion, owing to this pressure on the motor tract. Distinct evidences of hemiplegia, however, are not always observed. The condition of the pulse and arterial system is very much like that of ordinary apoplexy, but the respiratory system

is usually more seriously affected. Disturbances in the movements of the eyes and in swallowing, and in fact all those symptoms which show a pressure or irritation due to blood oozing into the fourth ventricle may be present. Death is almost sure to occur, and is inevitable if the hemorrhage, as is so often the case, breaks through and reaches the fourth ventricle.

Symptoms of the Chronic Stage.

Hemiplegia.—About two-thirds of the persons who are attacked with cerebral hemorrhage recover from the immediate effects. After passing through the symptoms that are connected with the attack they gradually improve, and at the end of from four to eight weeks enter upon what is termed the chronic stage of the disease. The patient is then commonly spoken of as a sufferer from hemiplegia or a "hemiplegic," since hemiplegia is the striking and important symptom in the case. The paralysis which had at first affected the whole of one side, so that the patient was perhaps barely able to move the arm and leg, has now become so much diminished that he can walk and use this arm somewhat, while the face seems almost entirely well. The paralysis, in almost all cases, disappears to the greatest extent from the face, so that it can only be detected by a close inspection of the facial movements, there being perhaps a slight diminution in the innervation of the lower muscles of the face, and the angle of the mouth on the affected side being a little lower than that on the sound side. Protrusion of the tongue will sometimes show a deviation slightly towards the affected side. The control of the orbicularis palpebrarum and muscles of the forehead is never much disturbed, and now shows no disturbance at all. In very old cases a certain amount of contracture may set in, so that the angle of the mouth on the affected side is drawn up, and when the patient laughs the face is pulled over towards the paralyzed side. This phenomenon is particularly frequent in hemiplegia occurring in children. The paralysis improves next in amount in the leg; the patient is able to stand upon the once paralyzed limb; he can easily extend the foot, but flexes it with more difficulty. Flexion and extension of the leg are less perfect, while the control of the thigh muscles is most limited of all, not because there is a great degree of paralysis, but on account of the stiffness of the part. The arm recovers least. The patient is able to swing the arm as a whole forwards and backwards, but he can raise it but slightly. He can flex the forearm and extend it with a fair degree of strength. The movements of the fingers and hand are imperfect. Flexion is fairly well preserved, and the patient can grasp things, holding a cane or a table utensil often with a certain degree of

ease; extension of the fingers, however, is very weak, and so also is supination, pronation being fairly well retained. It will be seen in examining cases of chronic hemiplegia that it is the extensor muscles of the toes and leg and thigh, also the extensors of the fingers, hand, arm, and upper arm, which are more involved.

The paralysis in both arm and leg is made very much more marked by the supervention of what is known as secondary rigidity and contractures. Both the rigidity of the muscles and the contractures are due to the gradual degeneration of the voluntary motor tract in the brain, and of the direct and crossed pyramidal tracts in the cord. The effect of this degeneration is to lessen the inhibitory activities of the higher cerebral centres, and as a result, an excessive amount of action is brought to play upon the flexor groups. As time goes on, what was at first simply stiffness and rigidity in the affected parts becomes a fixed condition of contraction, or, as it is more commonly called, contracture. These contractures affect, as I have said, the flexor groups, causing the toes to be drawn down, the heel to be elevated, and the foot extended, the leg to be flexed slightly upon the thigh, and the thigh slightly upon the trunk. In the arm the same process causes a tendency of the fingers to close upon the palms, of the wrist and forearm to be flexed, and of the upper arm to be held down next to the trunk. There is finally produced a condition of paralysis and contracture, which gives to the attitude and gait of the patient a characteristic appearance. He walks slowly, swinging the toe of the paralyzed side out, so that it scrapes the floor, forming the arc of a circle as he goes along. The arm is held to the side, the hands are closed, the body is also slightly bent forwards, and the position resembles that of senility. The muscles of the trunk are very slightly affected in hemiplegia, although in the early stages some disturbance may be noticed. This is due to the physiological law that all muscles which act synchronously upon symmetrical parts are innervated by each side of the brain. Thus the respiratory muscles of the chest and abdomen are innervated on each side by centres in each half of the brain, and when the centre in one cerebral hemisphere is destroyed the function is assumed by the centre on the other side. It has been shown, however, by recent researches that the pyramidal or motor tracts do not supply the opposite half of the body exclusively, but that some fibres go to the muscles of the same side. As the result of this, there is always in hemiplegia a certain amount of impairment of power even upon the sound side.

Along with the paralysis, rigidity, and contractures which I have described, there develops a condition of exaggeration of the deep reflexes. The patella tendon reflex, the triceps reflex, and the

Achilles tendon reflex are exaggerated. Ankle clonus can be obtained in most cases. During the acute stage of apoplexy the superficial or skin reflexes are often abolished on the paralyzed side, but in the later stages they reappear and become exaggerated also. Scratching the sole of the foot causes quick, jerking movements of the leg. Scratching the inner surface of the thigh produces a contraction of the cremaster, and so on.

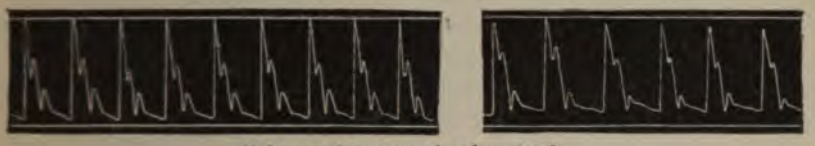
In a good many cases there is a slight amount of hemianæsthesia during the early stage of cerebral apoplexies, but this almost invariably disappears in a few days or weeks, and it is rare that any anæsthesia of the cutaneous or muscular senses is observed. Sometimes patients with hemiplegia suffer from pains in the affected parts. These pains are at times associated with cramp-like contractions of the arm or leg. In other cases they are burning, tearing, or neuralgic pains, and are due to the fact that the lesion irritates some part of the sensory tract or sensory centre in the brain. In hemiplegia of adults the affected side does not waste; the muscles, though but little used, do not become smaller to any notable extent. This is because the trophic centres for these muscles lie in the spinal cord—a part that is not affected in the disease. Once in a while, however, we find marked muscular atrophy in cerebral hemiplegia, though the cases are so rare that they may be considered freaks. When atrophy does occur it is, I think, usually due to the fact that the motor cortex is involved. In very bad cases of hemiplegia, in which the patient remains so paralyzed that he is for the most part bedridden, some atrophy takes place, though this affects, as a rule, both sides. In these cases also there may develop arthropathies—that is to say, a knee-joint, or an ankle, or an elbow may become enlarged and show evidences of exudation and later of degeneration of the osseous and connective tissues.

The patient's general bodily functions are usually carried on in a fairly normal manner. There is no great disturbance in digestion or in the activity of the kidneys. The sphincters of the bladder and rectum also perform their functions normally, although constipation is a frequent symptom, owing to the enforced inactivity of the patient. There is no doubt that the patient's general bodily vitality is somewhat lowered, and he is made somewhat more susceptible to the onset of infectious diseases or to the effects of renal, hepatic, or pulmonary troubles, which he may have had before the disease came on.

In almost all hemiplegics—in practically all who are past the age of forty—evidence of change in the arterial system is present. This is shown by hard, tortuous arteries, increased blood pressure, and the general signs of atheroma. In some cases, however, the athe-

roma and dilatation of the arteries is associated with a weakened condition of the heart. As a consequence of this, although the blood-vessels are dilated and stiff, yet they are not well filled with blood, and we get a characteristic sphygmographic tracing. In an article published in *The Post-Graduate*, 1893, p. 276, I gave several illustrations of this condition. (See Fig. 22.)

The mental condition of hemiplegic patients is a somewhat characteristic one. There is almost always a slight degree of mental impairment. This is shown in a weakness of memory and in an increase of emotionality, so that he laughs or cries easily and becomes easily excited or irritable, or easily depressed. In some cases the patients suffer from attacks of laughing or crying, which come on paroxysmally, with no exciting cause, or very slight, the patient be-



Male, age 68, two months after attack.

Male, age 56, four months after attack.

FIG. 22.—Sphygmograms of Radial Pulse Showing Arterial Dilatation and Low Tension after an Apoplexy.

ing thrown into spasms or uncontrollable laughter, which continue for many minutes or even hours. In this case, it is believed that the lesion has cut the fibres in the anterior part of the internal capsule, leading to the optic thalamus, which control the psychic reflex for emotional expression. He may suffer from insomnia. In many cases, however, the patient is able to pursue his former vocations, if they do not greatly tax his mental or physical powers.

A certain proportion of hemiplegics are affected in the beginning with disturbances of speech, which are known as aphasia. This aphasia is always associated with right hemiplegia in right-handed persons, and is due to lesions affecting the centres concerned in the faculty of language.

Among the rarer symptoms of the chronic stage of hemiplegia are certain mobile spasmodic movements of the affected side of the body. These spasmodic disturbances occur much more frequently in connec-

tion with hemiplegia in children than in hemiplegia in adults, but they are occasionally seen in the latter class of patients. One of the common disturbances is a slow rhythmical contraction of the fingers, hand, and arm, and also of the lower extremity. These contractions may be accompanied with pain; they are, in fact, muscular cramps due to the excessive irritability of the motor nerves and muscles of the paralyzed part. This form of spasm is known as athetosis (Hammond). Tremor is occasionally seen in the arm and leg. When present it is a tremor of the coarse type; that is to say, the rhythmical movements occur at the rate of five or six per second, and the tremor is one that is usually increased upon voluntary movement of the part. It rarely affects the face, but at times there is a certain amount of twitching of the tongue and facial muscles. Incoördinated movements of the hemiplegic side are also observed; the patient, on attempting to place the hand in a certain position, or to move it in a certain definite manner, goes through awkward and irregular ataxic-like motions. The condition is really an ataxia of the disordered limbs, and is due to an injury of the centres connected with the muscular memories. It is very rare indeed to find in adult hemiplegics anything in the nature of choreic movements. These, however, occur in infantile hemiplegia and even in hemiplegia occurring in youth; after the age of twenty, however, the hemiplegia is practically never associated with chorea. The same statements may be made with regard to the condition known as athetosis. Associated movements are sometimes observed in hemiplegics. By this is meant that when the patient tries to write, or makes some definite movements with the paralyzed arm or leg, there is at the same time a movement in the arm or leg of the sound side. The associated movements of the sound side resemble, in a measure, those of the affected side. The electrical irritability of the muscles in hemiplegia is at first somewhat increased; later it is diminished, but there are no qualitative changes in the ordinary types of the disease. Mirror writing is a phenomenon sometimes seen in hemiplegics, though more common in young children. A right-handed person who has right hemiplegia attempts to write with the left hand, and finds that he almost unconsciously writes from right to left, and reverses the letters so that in order to read them the sheet must be held before a mirror.

Pathological Anatomy.

The brain is supplied with blood chiefly by the internal carotids and vertebral arteries. The external carotids furnish blood to the

scalp, skull, and the dura mater. The internal carotids give off the anterior, middle, and posterior cerebral arteries, which join branches derived originally from the vertebrals, and unite to form the circle of Willis. They then send off arteries which supply the central ganglia and the pia mater of the cortex of the brain. The vertebral arteries supply chiefly the pons, the medulla, and cerebellum. The internal carotids furnish most of the blood to the cerebral hemispheres. The circle of Willis gives off sets of small, short branches, which pass up directly into the substance of the brain, supplying the basal ganglia, and these are known as the *central arteries*. The anterior, middle, and posterior cerebral arteries send off terminal branches, which supply blood to the pia mater and cortex, and they are known as the *cortical arteries*. Near their origin they also give off some central arteries.

Figure 21 (page 274) shows the brain laid open by a horizontal section, exposing the basal ganglia, and showing their relations to the internal and external capsules. The darkened area on the right side is that which is most frequently involved in cerebral hemorrhage, as well as in cerebral softening, more especially, however, the former. The parts injured, as will be seen, are chiefly the lenticular nucleus and the posterior portion of the internal capsule.

The constituents of this capsule have been described and figured differently by different authors. According to the latest investigations by Déjerine, the anterior portion of the capsule—*i.e.*, that between the caudate nucleus and the lenticular nucleus—is composed of fibres running from the frontal lobe into the anterior portion of the optic thalamus. At the bend or knee of the capsule is a small band of fibres, which come from the lower portion of the central convolutions (operculum Rolando), bearing, therefore, fibres that innervate the mouth, throat, and larynx. Behind this, and occupying the larger portion of this segment of the internal capsule, is a compact bundle of fibres that contains in its anterior part the voluntary motor tract, supplying the muscles of the opposite side of the body. Its posterior portion, reaching to the part opposite the putamen, contains fibres of muscular and articular sense. Behind this is a band of fibres, not so distinctly marked, which is known as the *bundle of Türck*. These come from the temporal lobe. Behind this bundle of Türck are fibres that come from the occipital lobe to enter the external geniculate body and the pulvinar of the optic thalamus. These are visual fibres.

In cerebral hemorrhage not due to trauma, one of the branches of the central arteries is affected in the vast majority of cases; hence intracranial hemorrhage not due to trauma is practically a hemor-

rhage into the central parts of the brain substance. The further away the blood-vessel is from the circle of Willis the less frequently do ruptures occur.*

In the common form of apoplexy due to cerebral hemorrhage, an examination of the brain discloses a clot of blood lying in the neighborhood of the corpus striatum, the internal and external capsules, and optic thalamus, sometimes breaking into the ventricles. The parts most frequently affected are, in their order, the external capsule and putamen, the internal capsule and corpus striatum, the optic thalamus, the centrum ovale. After this come in frequency hemorrhages into or upon the cortex, into the cerebellum and pons Varolii. Hemorrhages into the medulla oblongata, crura cerebri, and corpus callosum are practically never seen, except from trauma. The clot of blood varies in size from one-half inch in diameter to two or even three inches. Sometimes the central part of the hemisphere is hollowed out by a big clot, the size of an orange. In a majority of fatal hemorrhages the clot breaks into one of the lateral ventricles, and blood is found here; some of it oozes through into the third, and into the lateral ventricle of the opposite side. Occasionally the clot bursts through the cortex of the brain, and appears superficially in the subpial or even in the arachnoid space. Very rarely some hemorrhages appear primarily in one of the lateral ventricles, due to the rupture of some small artery or vein in the thalamus. Hemorrhages into the pons usually occur in the median line, and are generally small. Cerebellar hemorrhages are, as a rule, sufficiently large to cleave through the cerebellum and break externally, the blood flowing into the fourth ventricle. They are oftenest due to rupture of the superior cerebellar artery.

Cortical hemorrhages are usually small, being not more than half an inch or an inch in diameter. They may, however, extend along a fissure, such as the fissure of Rolando, lying deeply in it for a distance of two or three inches. These hemorrhages, when they occur primarily from rupture of cortical arteries or veins, lie beneath the pia-arachnoid membrane. They tear up and irritate the cortex, and may break through into the arachnoid space.

Hemorrhages that occur into the arachnoid space are due to a

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* The relative frequency with which different parts of the brain are affected with hemorrhage, as shown by my statistics, is as follows:

Hemorrhages involving the basal ganglia and ventricles	32
" " " meninges	14
" " " cerebellum	3
" " " pons	1
Total	50

rupture of one of the dural veins, or of the veins and arteries of the pia. These hemorrhages are often very large, covering the convexity of one hemisphere, and sometimes of both hemispheres. The clot may be half an inch or more in thickness. Such hemorrhages, when occurring idiopathically, are, as already stated, almost invariably the result of alcoholism or insanity. The clot in these cases is usually found to be composed of several superimposed layers, due to successive dural hemorrhages. The whole constitutes a condition known under the name of *pachymeningitis hæmorrhagica.*

The clot in the common form of apoplexy, if it does not break through into a ventricle, usually takes a somewhat ovoid shape, owing to a tendency of the blood to split away the longitudinal fibres of the centrum ovale and capsules. In some cases, instead of a distinct clot of blood, there will be found in the white substance or basal ganglia a collection of minute capillary hemorrhages, associated with softening of the tissue. This condition forms what is known sometimes as a *red softening.* It is seen oftenest in apoplexies which are complicated by Bright's disease or infectious fevers. In connection with these local changes one observes also that the convolutions of the brain upon the side of the hemorrhage are flattened, owing to the pressure of the extravasated blood. The meninges are generally congested, and often one finds an excess of cerebrospinal fluid, which may be tinged with blood. In addition to these changes, the blood-vessels at the base of the brain will be seen to be hard, grayish, and rigid, standing wide open when cut, owing to the existence of atheroma.

If life is preserved after a cerebral hemorrhage, certain reparative processes begin to take place: the blood coagulates, the serum separates out and becomes absorbed. This occurs at the end of about two weeks. During this time a fibrinous wall is formed, and a cyst gradually develops. This cyst, with serous contents and remains of the clot, may be noted by the twentieth or thirtieth day. The cyst gradually contracts, and, if the hemorrhage is small, there is finally left only a cicatrix.

In addition to this local change in the absorption of the clot and the production of the cyst, important secondary changes at once begin to take place in the nervous substance itself. Owing to the fact that hemorrhages so frequently occur in the neighborhood of the internal capsule, the voluntary motor tract, which carries impulses from the cortex of the brain to the motor cells of the spinal cord, is more or less completely cut in two. This motor tract is really made up of the nerve-cell processes (neuraxons) starting in the cortical motor area; and when these processes are cut off, their peripheral

portions die throughout their whole extent. Consequently, within a few days (from the tenth to the fourteenth) it is found that the whole of the motor fibres below the lesion, extending down into the spinal cord, begin to undergo a degenerative softening. The fibres ultimately are destroyed and disappear, and in their place connective tissue is developed. Hence, from the seat of the lesion, passing down through the crura cerebri and the pons and medulla and lateral and anterior median columns of the cord, there runs a strip of connective tissue instead of normal nerve fibres. This process is called a secondary degeneration, and its existence is the cause of many of the serious symptoms that are seen in the chronic stage following the apoplectic stroke.

The further morbid changes in the brain have to be made out mainly by microscopic examination. These all go to show that the fundamental trouble in cerebral hemorrhage is a disease of the blood-vessels. Much controversy still exists as to the exact nature of the changes. However, it can be stated with very considerable confidence that the following are the main pathological conditions:

First, a certain degree of atrophy of the brain substance itself.

Second, a degenerative change in the walls of the smaller arteries, and which may be included under the terms *arterial sclerosis* and *atheroma*.

Third, certain special changes which lead to the formation of minute aneurysms upon the terminal arteries, these being known as *miliary aneurysms*.

Fourth, specific disease of the blood-vessels, known as *endarteritis obliterans;* and

Fifth, various rare changes that may be included under the head of *amyloid degeneration* and *fatty degeneration of the arteries*.

1. Between the ages of forty and forty-five the brain begins to atrophy slightly, and as the result, the cranial space is less completely filled with brain tissue, its place being taken by cerebrospinal fluid. This fluid gives less support to the arterial walls than the brain tissue itself; hence this forms a factor, at least, although a minor one in dilating and rupturing vessels. The older pathologists attributed great importance to the atrophic process, and even considered that there was primarily a local softening or atrophy of brain tissue in the regions surrounding the broken vessel.

2. Arterial sclerosis and atheroma are included under the general name of *chronic vasculitis* or *chronic endarteritis*. The process affects chiefly those parts of the arteries at which branches come off, and it is due, according to Weichselbaum, to a diminution of the elasticity of the vessel wall, brought about by general disturbances of nutri-

tion, which are associated with advanced age, alcoholism, gout, and Bright's disease. As a result of this loss of elasticity the vessel dilates, the blood current slows up a little, and proliferation of connective tissue takes place in the internal coat or intima. In this way the lumen of the artery is made smaller again, or would be if a degeneration did not begin to take place in the arterial walls; but, owing to this degeneration, the new connective tissue of the intima becomes hyaline and fatty. It has little resiliency and the artery is dilated again. The connective-tissue proliferation now begins to affect also the middle and outer coats, and the degenerated parts of the newly formed tissues are filled with deposits of lime. In this way quite large calcareous plates are finally deposited in the arterial wall. This condition forms what is known as atheroma. The artery, as a final result, becomes a rigid, inelastic tube, that stands wide open when cut, instead of collapsing. During the process of this atheromatous change a blood-vessel may rupture, owing to the weakness of the wall produced by the degenerative process. Atheroma, however, of this type does not lead to actual obliteration of the arteries.

3. The presence of miliary aneurysms and their alleged great importance in the production of cerebral hemorrhage were first brought to the attention of the profession by the researches of Charcot and Bouchard, in 1872. These aneurysms had, however, been seen previously by Cruveilhier and Calmeil. Sir William Gull had also seen them, and in one case referred to them as a cause of cerebral hemorrhage. Charcot and Bouchard asserted that these aneurysms were practically always to be found in cases of idiopathic cerebral hemorrhage. They are usually not very numerous, but there may be more than a hundred. They are small in size, ranging from one-twenty-fifth to one-one-hundredth of an inch in diameter, and are fusiform or sacculated in shape. They are due, it is believed, primarily to a periarteritis, which leads to a weakening and dilatation of the walls. They involve only the very small arteries. The essential importance of miliary aneurysms in the production of cerebral hemorrhage has been maintained for many years, but later investigations do not entirely support the original view. Dr. Ludwig Stein, in the *Deutsche Zeitschrift für Nervenheilkunde*, Vol. VII., p. 313, has in particular controverted it. He states that he has not been able to find the miliary aneurysms in all cases, and that sometimes dissecting aneurysms, produced by the passage of blood into the lymph sheath of the arteries, result in appearances closely simulating aneurysms. He furthermore contends that the fusiform aneurysms may be found in perfectly healthy brains, and even in the brains of young people, and that they are not dangerous morbid conditions. Typical miliary an-

eurysms, he says, are present much less rarely than has been stated, and they play no great rôle in the production of brain hemorrhages, the most essential thing being the atheromatosis and syphilitic disease of the blood-vessels. Even Charcot and Bouchard admit that miliary aneurysms stand in no close relation to atheroma—that atheroma may be present without them, and *vice versa*.

4. Syphilitic disease of the blood-vessels. Syphilis exercises a selective influence upon the blood-vessels of the brain, owing to the fact that these vessels are supplied with lymph sheaths along which the specific poison travels. Among three hundred cases of brain syphilis, with autopsies, in which there were apoplectic attacks, in sixty-nine there was cerebral hemorrhage (Lechner). Syphilis leads to arterial disease, either directly by the production of an obliterative endarteritis or indirectly by the involvement of the blood-vessels in a syphilitic gummatous exudate or syphilitic node. In the former case, that of syphilitic endarteritis, the process resembles very much that described under the head of *atheroma*, only differing from it in that it does not terminate in fatty degeneration or calcification, but does lead to a narrowing and obliteration of the vessel. When the blood-vessels are involved through the proximity of a syphilitic node, all the coats are infiltrated; but the process seems primarily a periarteritis, with a secondary involvement of the other coats. During this process the vessel walls are finally obliterated. In some instances, however, during the process of the disease the wall ruptures and hemorrhage occurs. More often there is a blocking-up of the vessel, and thrombosis is the result. Syphilis, therefore, in only about one-sixth of the cases of apoplexy produces hemorrhage; in the other six, thrombosis. The vessels affected by the syphilitic process are chiefly the arteries at the base of the brain—namely, the basilar and its branches, and the circle of Willis and its immediate branches.

In addition to the foregoing forms of arterial disease, one occasionally finds fatty degeneration of the walls of the artery, due to septic states; and amyloid changes in the arterial walls.

Pathogeny.

The factors in the production of the cerebral hemorrhage are: a diminished support to the vessel wall, a diminished elasticity, weakness of these walls, and an increased arterial tension.

Of these factors, it is admitted that the most important, by all odds, is disease of the arterial wall. An increased arterial tension, hypertrophy of the heart, and abnormal peripheral resistance in the

capillaries play a minor part. It is probable that sometimes persons are born with peculiar anomalies of the arteries which render hemorrhage more possible, as, for example, abnormally small brain arteries in otherwise well-developed arterial systems.

The reason for the particular location of cerebral hemorrhages is explained upon simple mechanical causes. The blood pressure in the internal carotid is about one hundred and fifty millimetres. Now, the blood passing through the internal carotid goes almost directly into the circle of Willis, and with almost equal directness into the central arteries which are given off from it and its branches; consequently the blood pressure in these central arteries is relatively high. On the other hand, the blood pressure in the cortical arteries, owing to their length and wide distribution, is much lower. One of the arteries which seems to receive this excessive pressure most directly is a branch of the middle cerebral, known as the *lenticulo-striate artery*, and Charcot calls this "the artery of cerebral hemorrhage."

Diagnosis.

In the diagnosis of hemorrhagic apoplexy we have a number of problems to consider:

I. To distinguish hemorrhagic, embolic, thrombotic, and hysterical apoplexy from other conditions.

II. To distinguish hemorrhagic apoplexy from embolism, thrombosis, and hysteria.

III. So far as is possible, to locate the position and extent of the hemorrhage.

When one is called suddenly to a patient who has fallen down unconscious he must keep in his mind the possibility of the following conditions being present:

Hemorrhage, thrombosis, embolism.

Drunkenness.

Concussion and shock from some injury to the head.

Syncope.

Some form of hysteria or epilepsy.

Uræmic or diabetic coma.

Opium poisoning or poisoning by other narcotics.

As a matter of practical experience, we know that most of these cases of sudden unconsciousness which a physician is called to attend are due to apoplexies, epilepsy, hysteria, alcohol, or concussion of the brain. In many cases we know, too, that a few questions from the friends or relatives very soon inform the practitioner of the nature of the trouble without a very thorough examination on his part.

It is seldom that a case of alcoholism occurs without a pretty distinct previous history associated with it. When a person falls into an epileptic coma, there is usually some previous history that throws light upon it also; and so with the other comatose states to which the physician is called. I need only suggest a few practical points which may be borne in mind.

In cases of apoplexy, for example, whether due to hemorrhage or to acute softening, there rarely fails to be some evidence of the hemiplegia. This can be brought out either by pricking the palms of the hands or the soles of the feet on the two sides, or by lifting the limbs and seeing how they fall. In severe forms of apoplexy with hemorrhage in the ventricles, there is sometimes stiffness, even at an early period, of the affected side. In addition to that, in apoplexy the pupils are rarely even, while in other conditions they are usually so. The presence of some heart murmur of very marked character may at once suggest that there is embolism. On the other hand, the stertorous respiration, the slow, hard pulse, the flushed face, together with the physiognomy and age of the patient, may show at once that it is a case of cerebral hemorrhage. In cerebral hemorrhage, if there is coma the patient cannot easily be aroused, and even if aroused may be aphasic and unable to understand and answer questions. In most of the other comas vigorous excitation of the patient will usually bring out coherent words of some sort. This is particularly the case with the coma of alcoholism and apparent coma of hysteria. If there is a suspicion of the hysterical condition, the use of cold affusions, of pressure on the supraorbital nerve, or an emetic will often break up the attack. It not infrequently occurs in this city that acute alcoholism is associated with concussion of the brain, and sometimes even with hemorrhage. Patients brought to the hospitals in this condition often present symptoms that it is very difficult to analyze, and I do not believe it is always possible at once to distinguish alcoholism associated with concussion, due perhaps to a blow on the head in some fight or fall, from a hemorrhage in the brain. As a general rule, we know that in acute alcoholism the patient is not absolutely comatose, but can be aroused slightly. His pupils are even, he has no hemiplegia, his breath is strong with alcohol, he has no convulsions, he often struggles violently, and he presents in general the physiognomy of a drunken man, which is familiar to the hospital and general practitioner.

The coma of uræmia is, in my experience, a relatively rare condition, unless we consider the coma which simply precedes death. Ordinary attacks of uræmic coma are certainly rare in hospital experience, and twitchings of the muscles and mild convulsive attacks are

much more frequent manifestations of uræmic poisoning. Uræmic coma usually comes on slowly, but if it does occur suddenly, as it may do, it is easily distinguished by the manifest evidences of Bright's disease obtained from examination of the urine, by the physiognomy of the patient, the equal pupils, absence of hemiplegia, and the tense condition of the arteries. The patient can usually be aroused. The pupils are, as a rule, rather contracted than otherwise. Rarely they are finely and evenly contracted, but they may be dilated. The respirations are not stertorous, and are oftener accelerated than retarded. The coma of syncope and the coma from concussion of the brain can usually be recognized by the history of the case and the examination of the heart and of the head. The temperature is usually normal or lowered.

The coma of opium poisoning is recognized by the history of the case and presence of slow breathing. It is not of the Cheyne-Stokes type, and is not stertorous, but is simply very slow, being reduced sometimes to three or four a minute. The face is slightly congested, the skin often moist, and there may be a slight rise of temperature, from one-half to one degree; the pupils are contracted to a pinpoint; the patient cannot, as a rule, be roused at all. There is no evidence of hemiplegia, no twitchings or convulsive movements, the pulse is not slow and hard, but rather rapid. In some cases hemorrhage into the pons produces a condition of symptoms closely resembling this. The history of the case, the fact that sometimes the patient vomits, and the examination of the contents of the stomach, obtained by a stomach pump, will, as a rule, usually settle the diagnosis.

In hysterical apoplexy the coma is not profound, the face is not involved in the paralysis, there is usually total hemianæsthesia, and later contraction of the visual field, hemianopsia, hemiageusia, and bone deafness on the affected side are to be noted.

To sum up, the physician who is called to make a diagnosis of the comatose patient should observe the following rules:

Get the history of the patient as completely as possible. Note the general physiognomy of the patient and bear in mind the impression which that first glance gives, for to the experienced observer there is nothing so valuable as the instinctive judgment which comes to one when first brought into the presence of disease. Begin a careful examination of the different functional disturbances of the patient. Examine the mental condition, the depth of unconsciousness that exists. See if the patient can be roused by calling or by any kind of stimulation. Examine the heart for evidence of disease or of failing power. Examine the pulse for its rapidity, its regularity, and tension. Note the character of the respirations, particu-

larly whether they are slow, and whether they are of the Cheyne-Stokes type. Note whether there is any paralysis, either in the form of hemiplegia or otherwise. Move the limbs in order to test their flexibility. Test the patella tendon reflex. Note with the help of a pin or some instrument causing irritation, whether there is anæsthesia or an exaggeration of the skin reflexes. Examine the pupils, their equality, their reaction to the light.

Take the temperature of the body, and if apoplexy is in any wise suspected, take the temperature of each axilla, as well as in the rectum, in order to determine the relations of the temperature between the two sides of the body. The head should always be carefully examined to distinguish whether there are any evidences of bruises and fracture. The ears should be examined for signs of rupture of the drum or hemorrhage. The tongue should be examined, in many cases, to see whether there has been any biting of it during convulsions. Finally, the stomach contents may have to be tested for poison, and the urine to determine the question of uræmia.

The second problem in diagnosis is to distinguish between hemorrhage, embolism, and thrombosis.

Practically the distinction lies most frequently between thrombosis and cerebral hemorrhage. Cerebral hemorrhage occurs most frequently in the middle periods of life, between the ages of thirty and fifty. After the age of sixty-five or seventy and before the age of thirty and thirty-five we should expect softenings from senile or syphilitic arteritis. There is in hemorrhage often a history of arterial disease, that is to say, of gout or chronic alcoholism, or Bright's disease. We have also sometimes an hereditary history of cerebral hemorrhage which may help us in diagnosis. Hemorrhage has, as a rule, few marked premonitory symptoms; the patient may even be feeling unusually well at the time of his attack, and a history of this condition gives rise to a strong presumption of vascular rupture. Cerebral hemorrhage is sometimes, though not always, the result of a sudden exertion, whereas thrombosis rarely occurs under such a condition. Hemorrhages occur more often in the morning or evening, and they do not often occur during sleep. When, therefore, a person wakes up in the morning and finds that he has a hemiplegia we may pretty safely assume that it is due to an acute softening. Cerebral hemorrhages come on suddenly and usually with loss of consciousness. The flushed face, the slow, hard pulse, the throbbing carotids, and other evidences of intense cerebral congestion and overaction of the heart, all point towards hemorrhage. On the other hand, in thin, anæmic, and weakened individuals the occurrence of a sudden hemiplegia would be more likely to suggest an acute softening. In cerebral

hemorrhage consciousness is more frequently and profoundly lost, while in acute softening the patient may retain consciousness or be only semicomatose. An inequality of the pupils, indicating an unequal pressure in the brain, suggests hemorrhage rather than softening. Vomiting indicates hemorrhage rather than softening; but convulsions may occur in either state.

In cerebral hemorrhage, if it is at all large and there is decided shock with loss of consciousness, the temperature in the rectum within a few hours after the attack sometimes falls two or three degrees. After twelve or twenty-four hours the temperature in cerebral hemorrhage is apt to be a little elevated, perhaps about one degree, and usually it is a degree higher on the paralyzed side; on the other hand, in acute softenings the temperature of the body is very slightly affected; it is equal or nearly so on the two sides, and practically normal in the rectum. In hemorrhages also the temperature is more apt to rise a little on the second or third day, while in acute softening such change does not occur unless the softening is very great, or affects the pons, or is associated with some septic process. In cerebral hemorrhage one finds more evidences, as a rule, of arterial disease than in embolism, but rather less than in thrombosis, unless the patient be a syphilitic. A history of syphilis, especially of an infection occurring within three or four years, suggests a thrombosis and softening rather than a hemorrhage, although sometimes a cerebral hemorrhage does occur as a result of syphilis.

Finally a quicker return of consciousness and a more rapid progress towards recovery indicate softening rather than hemorrhage. Hemorrhages are more serious and more fatal than softenings. Embolism practically never occurs without a serious and generally an active form of endocarditis being present. It does, however, occur after pregnancy and in certain cases of sepsis. When, therefore, a person has an abscess in any part of the body or an acute endocarditis the possibility of embolism may be considered great. The age of the patient is also a help. Embolism rarely occurs in the aged, and is most frequent in the earlier periods of life, at least before the degenerative changes have set in. The history of syphilis would lead one to make a diagnosis of thrombosis or hemorrhage rather than embolism. Embolism occurs more often in women, and is rare in children. A profound anæmia would lead one to expect an embolism rather than thrombosis, though this factor has not a very great weight. In embolism one rarely gets a history of any premonitory symptoms, while in thrombosis a previous history of headaches, mental confusion, vertigo, paræsthesia, and transitory paralysis is often found. The onset in embolism is always sudden; it begins

often with some motor symptoms, that is, a slight convulsive twitching or a slight hemiplegia followed by complete paralysis. In thrombosis the symptoms come on gradually.

The rules for the localization of the lesion must be drawn from the symptoms given under the heads of the different clinical types of apoplexy.

Prognosis.

The prognosis of intracranial hemorrhage depends a great deal on the seat and size of the lesion. Taking intracranial hemorrhages as a whole, it is my experience that one-half to two-thirds recover from the first attack of hemorrhage, less than one-half of the remainder recover from a second attack, and very few indeed recover from a third; although I have known persons to have as many as five successive attacks, dying in the last. Apoplexies in which there is a very profound loss of consciousness, lasting for at least three days, are usually fatal. If the temperature, instead of falling on the second or third day, begins to rise, the outlook is uniformly unfavorable. The appearance of Cheyne-Stokes respiration is an extremely bad sign, although I have seen a case in which recovery took place after a free venesection. When any symptoms develop during the attack which indicate involvement of the medulla the prognosis is most unfavorable, and the same may be said of hemorrhages involving the tubercula quadrigemina and causing ocular paralysis. When a case of apoplexy which is apparently doing well suddenly develops coma or convulsions, the outlook is most unfavorable, for it indicates the extension of the hemorrhage. Cases in which the hemiplegia is very complete, so that the patient can move neither hand nor foot, are most serious, though not necessarily fatal. On the other hand, when the attack is not associated with loss of consciousness or rise of temperature to any marked degree, the outlook is very good. The presence of Bright's disease is of bad omen, and if pneumonia develops the patient dies. It is difficult to prove it, but it seems to me probable that, other things being equal, hemorrhages are less fatal with young persons than they are with the aged. This would follow naturally from the fact that the brains of the aged are not so well nourished, and that the tissues present less resistance.

The presence of diabetes, chronic alcoholism, a convalescence from typhoid fever, idiopathic anæmia, furnish unfavorable conditions. In many cases cerebral apoplexy seems to put a check upon the activity of the patient, enforcing a quiet and regular life, and tending even to prolong existence. It may be thus considered almost a conservative process. Many cases of hemiplegia live for five or

ten or more years. The prognosis as to the recovery from the paralysis also varies very much. As a rule, recovery begins to take place within a fortnight, and it continues up to the end of a year. Rapid improvement, however, ceases at the end of a couple of months, and after that time progress, if it takes place, is very slow.

Treatment.

The treatment of the condition depends naturally very much upon the fact whether or not the physician has been able to make a positive diagnosis of intracranial hemorrhage. If he is satisfied upon this point prompt and vigorous treatment may be of great use. The patient, if seen early, should be placed in bed, with the head and chest well raised and the clothes loosened, so that the circulation from the head is not impeded. The extremities should be swathed in hot cloths wrung out in mustard water; an ice-bag should be placed upon the head; a drop or two of croton oil mixed with a little sweet oil should be placed upon the tongue. It may sometimes be a good practice in cerebral hemorrhage to attempt compression of the carotid upon the affected side, provided the patient is seen within two or three hours of the attack, and the symptoms do not show positively that there is a rupture into the lateral ventricles. If this has occurred, carotid compression can avail little. Compression should be continued for three-quarters to one hour. The suggestion which has been made to tie the carotid is not to be recommended. To relieve the stertor, turn the patient on the paralyzed side and see that the tongue is drawn forwards. In plethoric patients with a strong heart action, congestion of the face, and strong evidences of great cerebral hyperæmia and overaction of the heart, bleeding to the amount of ten or twelve ounces is advisable, and I have seen some desperate cases apparently brought up by this measure. Contrary to what might be expected, bleeding, if not done to great excess, does not necessarily injure a person with thrombosis. I base this statement on cases in which thrombosis has occurred, bleeding has been performed, and symptoms of improvement have appeared. I would not have it understood, however, that I consider it at all advisable to bleed in such conditions, but rather the contrary. If the patient's condition is such that one does not feel justified in bleeding, a somewhat similar effect can be produced by giving two or three drops of tincture of aconite every half-hour until the evidences of lowered arterial tension and weaker heart action are obtained. The tincture of veratrum viride is recommended for the same purpose. Duquesnel's aconitine in a dose of gr. $\frac{1}{200}$, repeated in three hours, may

be used instead of tincture of aconite, which is not always a trustworthy preparation. The administration of ergot has been advised, but I do not believe in its utility, nor have I seen any great results from bromides. The preparations of hydrastis are recommended strongly as useful in hemorrhage; but their value in cerebral hemorrhage has, so far as I know, not yet been determined. In some cases of cerebral hemorrhage the shock and the weak condition of the patient are such that there is evidence of heart failure, and if this be present then we must not hesitate to use small amounts of alcohol, digitalis, and strychnine.

After the first few hours any attempt to control the hemorrhage is futile; it has by this time done its work. There may, however, sometimes be a second rupture of an artery or a breakage into the ventricles, and in order to avoid this the greatest care must be taken to keep the patient extremely quiet. If he is restless and delirious, bromide and chloral, or morphine, should be given, preferably the former. On the second or third day it is advisable in suspected syphilis to give small doses of iodide of potassium; that is to say, doses of two or three grains every two hours. If the patient gradually recovers consciousness all medication may be suspended except the iodide of potassium and such measures as may be indicated in accordance with the development of the symptoms. At the end of ten days or a fortnight, if the patient has satisfactorily improved, one may begin cautiously to apply the faradic current to the paralyzed limbs. If the paralysis is slight this need not be done so soon; but if complete, there is some benefit in beginning electrical applications early, provided they are made very short and very mild; that is to say, each group of muscles should be made to contract three or four times by means of the current. If a patient does not progress favorably, if he continues in a state of partial unconsciousness and develops a slight fever, there is little to be done except to keep the emunctories open; the skin should be bathed with warm water, the kidneys be acted upon by sweet spirits of nitre and iodide of potassium, and the bowels should be kept regular. A milk diet is to be recommended during this time. Special care should be taken lest pneumonia develop. The mouth should be kept thoroughly disinfected, and the patient should be prevented from lying too long in the same position. Thorough cleanliness is necessary in order to prevent the development of bedsores, for in some cases the patients void the urine and fæces in the bed.

When the chronic stage has been reached the medical treatment will have to vary very much in accordance with the general health of the patient. In some cases in which there is evidence of syphilis

large doses of iodide of potassium, with occasional courses of mercury, must be given. If the patient is gouty and has some renal complications the use of tincture of iron and of the iodides, or the acetate of potash and digitalis, may be indicated. Strychnine is of some use, not because it directly affects the paralyzed limbs, but because it strengthens the heart and is a good general tonic.

For the first year after his stroke the patient should have courses of electrical treatment, massage, hydrotherapy, and mechanical treatment of various kinds. An electrical treatment should not last more than six weeks, and, as a rule, three applications a week are sufficient. The faradic battery with the current of high tension is a useful one, but it seems from my experience that the long sparks of the static machine give the most successful results. I do not know that any advantage is to be gained from the use of galvanism. After a course of electrical treatment the patient may rest a week, and then have a course of massage daily for a month. He should rest then, and may afterwards begin the electricity again. In some instances lukewarm baths are useful auxiliaries. The patient should be placed in a bath of a temperature of 95°, and should be made to exercise his muscles while there for a period of about ten minutes daily. He should afterwards be taken out and rubbed well with cool water and alcohol. In some patients the contractures of the affected limbs are very great, and the deformities resulting render the limbs almost useless. The fingers of the hand are particularly apt to be affected in this way. I have, in some instances, had the arm and the foot hyperextended and placed in plaster-of-Paris with fairly good results. It has seemed to me that if a patient began his treatment by steady attention to the prevention of the excessive contracture a good deal of it might be avoided. Some improvement in the hemiplegia may be expected for over a year, after that not much can be done; still in cases which have not been thoroughly treated in the first year, or in cases in which there has been neglect of mechanical treatment, help may be given even late in the disease.

Acute Softening of the Brain—Embolism and Thrombosis.

Acute softening of the brain is a condition caused by the plugging of a blood-vessel with an embolus or thrombus, and is characterized by a more or less sudden apoplectic seizure; the symptoms in the later stage resemble those that follow intracranial hemorrhage.

Etiology.

Embolism occurs rather more often in women, thrombosis in men. Embolism is rare in children, being most frequent between the ages of twenty and fifty; thrombosis occurs oftenest between the ages of fifty and seventy. The most important predisposing factors in embolism are acute or recurrent endocarditis, infectious fevers, profound anæmia, pregnancy, and blood dyscrasiæ; in thrombosis, syphilitic, lead, or gouty arteritis, fatty heart, and blood dyscrasiæ. The same causes which lead to the arterial disease that produces cerebral hemorrhage also predispose to thrombosis, though in the latter condition atheroma and syphilitic arteritis play the important part.

Symptoms.

In embolism there are rarely any premonitory symptoms; the onset is sudden; it may begin with some convulsive twitchings, then follow hemiplegia and temporary loss of consciousness. Coma, however, is rarer than in hemorrhage, and if present is usually shorter. There is rarely vomiting, nor do we find the hard, pulsating arteries, flushed face, and severely stertorous breathing. The initial temperature changes are slight, but in a few days fever may develop.

In thrombosis premonitory symptoms are frequent. In syphilitic cases there are headaches and cranial nerve palsies. In other cases vertigo, temporary aphasia, transient hemiplegia, numbness of the hand and foot, and drowsiness may be present. The onset is more gradual; the hemiplegia slowly develops, taking several hours, perhaps, for its completion; meanwhile the patient gradually becomes comatose. The attack sometimes is rather sudden, with no loss of consciousness, and it may occur in sleep. The temperature often has a slight initial fall, followed by a rise, just as in hemorrhage. In both embolism and thrombosis the hemiplegia tends to improve very much in a few days or weeks unless the vessel obliterated is a large one.

In embolism the right side of the body is affected slightly more than the left, owing to the fact that the left middle cerebral is rather more easily reached by an embolus. Thrombosis affects the two sides about equally.

Acute softening may kill within twenty-four hours; but, as a rule, the patient survives the onset, and if he dies it is not for several weeks. After the acute stage is over the patient passes into the

chronic stage, which resembles in nearly all respects that of hemorrhage. (See page 278.) After an acute softening, however, it is believed that there are more spastic symptoms and a greater tendency to mobile spasm. In embolism, owing to the youth and freedom from arterial disease, the mind is less affected; while in thrombosis the contrary is the case.

Prognosis.

The prognosis as regards the attack is somewhat better than in hemorrhages as a rule. In embolism it is good as regards recurrence; in thrombosis, bad. The mental condition is better in embolism; usually worse in thrombosis. The recovery from attack is more complete in acute softening. After the chronic stage is reached, however, the prognosis is about the same in all forms.

Diagnosis.

The diagnosis between acute softening and hemorrhage has been sufficiently dwelt upon under the head of *cerebral hemorrhage*. The diagnosis of embolism is chiefly made out by the suddenness of the attack, the age of the patient, the presence of heart disease, of pregnancy, or the puerperal state. The principal factors in the diagnosis of thrombosis are, the advanced age of the patient, or, if the patient is young, a history of syphilis, the presence of premonitory symptoms, and the gradual onset of the attack. The absence of disturbance of temperature is in favor of acute softening, rather than hemorrhage.

Pathology.

The embolus or thrombus cuts off the blood supply from a certain area of brain tissue. In twenty-four hours this begins to soften. If the area is in the cortex it becomes red (red softening); if in the white and less vascular part, it is usually white with a few red punctate spots. The red softening gradually becomes yellow (yellow softening). The dead tissue softens and is absorbed, leaving a cicatrix or cyst. If the embolus contains infective microbes there may be a local encephalitis and abscess.

In thrombosis there are usually evidences of extensive atheroma or of syphilitic arteritis. In those instances in which the thrombosis is caused by the blood state, as in scurvy or after wasting fevers with a weak heart, little arterial change occurs. Atheroma affects chiefly

the internal carotids and the large arteries at the base, viz., the middle, anterior, and posterior cerebrals and the basilar and vertebrals.

Thrombotic softenings occur oftenest in the corpora striata and optic thalamus, next in the pons and medulla. Thrombosis affects the vertebrals, the basilar, and the posterior cerebral arteries much oftener than do hemorrhages or embolism. An embolism may, however, plug up the basilar or the vertebral at its junction with the basilar. Embolism usually affects the middle cerebrals (seventy-five per cent. of seventy-nine cases, Pitt) and there is a preponderance in favor of the left side. The cerebellum is practically not affected by embolism or thrombosis.

As a guide in determining the location of the artery affected in thrombosis, the following facts are given, taken mainly from Birch-Hirschfeld:

Softening in the region of the frontal convolutions on the inner surface of the hemispheres as far as the callosomarginal sulcus—due to obliteration of the trunk of the anterior cerebral artery.

Total softening of the territory of the artery of the fissure of Sylvius, including the corpus striatum—due to obliteration of the first two centimetres of this artery.

Total softening of the cortical areas supplied by the artery of the fissure of Sylvius, the corpus striatum excluded—due to occlusion of the artery just beyond the giving-off of the arteries of the corpus striatum.

Partial softening in the area supplied by the arteries of the fissure of Sylvius—due to embolism of the inferior external frontal artery. The result is a softening of the island of Reil and of the third frontal convolution. If this is on the left side, aphasia results.

Softening in the posterior part of the second frontal convolution and in the first central convolution—due to obliteration of the anterior parietal artery.

Softening of both central convolutions and the Rolandic fissure, the anterior part of the first parietal convolution, and the island of Reil—due to closure of the median parietal artery.

Softening of the lower parietal convolution and the first temporal convolution as well as of the island of Reil—due to obliteration of the posterior parietal artery.

Softening in the area of the posterior cerebral artery, rarely complete, involves the inferior part of the occipital lobe and the tip of the occipital lobe.

Total softening of the corpus striatum, including the capsule, the lenticular and caudate nucleus and the anterior third of the thalamus

—seat of thrombus in the beginning of the artery of the fissure of Sylvius.

Partial softening in the form of a cone whose apex lies in the anterior part of the lenticular nucleus, while the base, directed forwards, involves the anterior two-thirds of the corpus striatum; the cone is formed by the anterior part of the nucleus caudatus, the internal capsule, and the third segment of the lenticular nucleus—the seat of the occlusion here lies in the lenticular striate artery.

Partial softening lying more posteriorly than the preceding, involving the posteroexternal part of the lenticular nucleus, the internal capsule, the anterior part of the thalamus, and the tail of the corpus striatum—the lesion involves the lenticular optic artery.

Partial softening of the thalamus opticus, the lesion ranges in size from a pea to a hazelnut—the vessels involved are the perforating arteries of the choroid plexus.

Softening of the frontal, parietal, and sphenoidal lobes—due to embolism at the point of bifurcation of the internal carotid, extension of the thrombus into the anterior cerebral artery.

Treatment.

The treatment of the attacks consists essentially in rest and such attention to the bowels, kidneys, and heart as may be indicated. In thrombosis it is important to give heart stimulants and arterial depressants, and for this purpose I advise the use of alcohol, digitalis, or strophanthus with nitroglycerin. Iodide of potassium and mercury ought to be given if there is the slightest suspicion of syphilis. Later one should prescribe courses of the iodides and mercury and of strophanthus, nitroglycerin, strychnine, and such tonics as may be indicated. The symptomatic treatment of the chronic stage is the same as in hemorrhage.

TUMORS OF THE BRAIN.

BY

B. SACHS,

NEW YORK.

TUMORS OF THE BRAIN.

THE study of tumors of the brain is of the greatest importance both from a scientific and from a practical point of view. The clinical manifestations of cerebral neoplasms resemble closely those of other intracranial diseases, such as abscesses, chronic forms of meningitis, etc. Consequently, the consideration of brain tumors calls for careful differentiation between the symptoms produced by them and by the other morbid processes. The attempt to locate the new growths within the cranial cavity has also given a great stimulus to the accurate determination of the cortical areas. More than this, the successes and the failures of cranial surgery have lent a peculiar interest to the subject in hand. The advisability of attempting the removal of intracranial growths is at the present time under serious discussion both here and abroad. Taking all this into account, it is not surprising that the subject has been considered in all its bearings by a number of competent writers. Among them we mention the names of Bernhardt, Bramwell, Gowers, Mary Putnam Jacobi, Knapp, Mills and Lloyd, Peterson, and Starr.

The latest monographs by Bruns and Oppenheim contain excellent critical summaries of the main questions at issue and are models of their kind. It would be futile to attempt once more to go over the literature presented by these authors. The present writer will refer to some of the cases reported during the past two years, but his chief aim will be to give a succinct presentation of the entire subject, with such additions as a large personal experience has suggested. Mere statistical inquiries are not needed. In this as in so many other subjects, figures may prove too much or too little. Inferences drawn from a few well-observed cases are worth more than those from dozens of others in which the results were not properly analyzed.

DEFINITION.

Tumors of the brain are to include solid new growths upon or within the substance of the brain. Abscesses are excluded from consideration, except that they will have to be mentioned in the section on differential diagnosis; aneurysms, however, give rise to the same

symptoms as intracranial tumors, and will be referred to as though they were solid tumors. Nor can cysts be excluded, since a rapidly growing cyst simulates the manifestations of solid tumors so closely as to be practically indistinguishable from the latter. Not infrequently, too, neoplasms issue from the walls of an old cyst.

Occurrence.

Tumors of the brain may be developed during any period of life. They are least frequent in the period of extreme senility and in the first years of life. Carpenter has lately reported a large tumor in a child of four months. Children under ten are relatively more liable to these diseases than youths between ten and nineteen (according to Starr's tables in Keating's "Cyclopedia"); but the meaning of such statistics can be gauged easily if we remember that one hundred and fifty-two of three hundred cases in children (collected by Starr) were of a tuberculous character and such hereditary disorders are apt to be developed in the first years of life.

Between the ages of twenty and fifty every form of tumor may occur. While tubercles and cystic tumors are relatively more frequent in early life, gliomata, sarcomata, and gummata are relatively more common in adults than in children. These facts are based upon the following tables taken from articles by Starr.

In spite of the large number of cases that have been recorded by various writers the actual frequency of tumor of the brain is far greater than such figures would indicate. Hundreds of cases have been observed which have not entered into the literature of the subject. I have notes of at least fifty cases, and only a few of these have been referred to in my previous writings.

TABLE I.

(Somewhat condensed.)

Age at which Tumors are Found.

Under one year	7 cases.	From ten to twelve years	33 cases.
One year old	8 "	Thirteen years old	8 "
Two years old	19 "	Fourteen years old	13 "
Three years old	17 "	Fifteen years old	10 "
Four years old	22 "	Sixteen years old	3 "
Five to seven years old	50 "	Seventeen years old	14 "
Eight years old	7 "	Eighteen years old	12 "
Nine years old	13 "		
Total under ten years	143 cases.	Total between ten and nineteen years	93 cases.

TABLE II.

(The first column are children's tumors; the second column adults' tumors.)

Situation.	Tuberculous.		Sarcomatous.		Gliomatous.		Gliosarcomatous.		Cystic.		Carcinomatous.		Gummatous.		Other varieties.		Total.	
I. Cortex cerebri	13	9	1	46	6	19	..	8	..	..	1	19	..	13	..	12	21	127
II. Centrum ovale	6	2	5	7	1	11	1	4	15	..	1	3	1	..	5	4	35	51
III. Cerebral axis:																		
1. Basal ganglia and lateral ventricles	14	3	5	8	3	9	..	..	1	1	1	2	..	1	3	5	27	34
2. Corpora quadrigemina and crura cerebri	16	1	3	2	1	2	..	5	..	..	..	1	..	..	1	7	21	14
3. Pons	19	11	5	1	10	..	2	1	1	..	..	2	..	3	1	..	38	17
4. Medulla	2	..	..	..	..	1	..	..	1	..	2	..	..	..	1	..	6	2
5. Base	..	3	1	3	..	2	1	1	1	..	1	..	..	..	4	1	8	9
6. Fourth ventricle	1	..	1	1	1	..	..	..	..	..	1	2	..	..	1	1	5	4
IV. Cerebellum	47	8	10	13	15	8	1	6	9	..	3	..	..	..	11	10	96	45
V. Multiple tumors	34	4	3	5	..	2	..	..	2	1	..	2	1	3	3	1	43	17
	152	41	34	86	37	54	5	25	30	2	10	31	2	20	30	41	300	300

Etiology.

It is quite as difficult to account for the occurrence of cerebral tumors as for those situated in other organs of the body. The origin of the infectious and parasitic forms (tubercles, gummata, echinococcus cysts) is evident enough. The large brain cysts, though often found in adult brains, are to be traced to early vascular accidents or to early encephalitis. Carcinomata are rarely, if ever, primary in the brain; they as well as sarcomata are metastatic in origin. Oppenheim thinks that there is a possible relation between the development of sarcoma and the previous existence of syphilis. The unusual frequency of glioma is explained in part by the tendency of cerebral tissue to proliferate, and this is especially true of the infantile brain; in part also on the theory of Virchow and others, that gliomatous tumor formation is due to the survival of abnormal embryonal deposits.

Traumatic injuries to the skull have been held to be a powerful etiological factor. Starr thinks that the falls of children upon the occiput account for the relative frequency of cerebellar tumors in them; but in all probability the accident serves merely as a landmark in many of the cases, for it would be difficult to tell why tumors of such varying histological character should be developed after skull injuries. We can conceive of a direct causal relation between a traumatic injury and the development of cysts, of fibroma, of osteoma, and of glioma, inasmuch as the injury may have caused the embryonal tissue to enter upon a process of proliferation (Oppenheim), but why tubercles and sarcomata should occur after injuries we cannot as yet

explain. Wernicke attaches some importance to heredity, at least to a family predisposition to nervous diseases, but I have not yet seen a single instance of the occurrence of tumor of the brain in successive generations, and I am certain that few such have been recorded.

We may now pass to a short account of the

Morbid Anatomy.

Glioma is generally a primary formation; its occurrence after glioma of the retina has been described by Arnold and v. Recklinghausen and referred to by Oppenheim. Glioma is characterized by the same elements which are to be found in neuroglia tissue. The

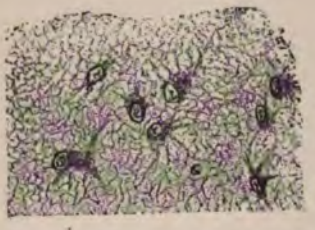

Fig. 23.—Hard Glioma from a Child's Brain, × 400, showing numerous cells among the fine network of fibrils in the neuroglia. Figs. 23-29 after Ribbert.

Fig. 24.—Glioma of Brain, showing oval nuclei forming central points for the arrangement of the fibrils.

main tissue consists of a dense network of delicate fibrils; some, though by no means all, of these fibrils are the ramifying processes of small cells (Figs. 23 and 24). These cells are distinguished from others by the presence of round or oval nuclei. If there is a relative preponderance of fibrous tissue, the consistence of the tumor may be changed into that of a fibroglioma. Klebs described a variety of glioma in which there is an excess of nerve elements, and for this special formation proposed the term neuroglioma. The character of a gliomyxoma can be inferred easily from the composition of the name.

The ordinary glioma is at times sharply differentiated from the surrounding cerebral tissue; at other times, particularly in those tumors occurring in the vicinity of the pons, the gliomatous tissue does not stand out prominently above the surrounding tissue. It is often more correct to speak of a gliomatous infiltration than of a distinct new growth. The new growth in the cortex is at times so like the surrounding tissue that after exposure of the cortex the surgeon

and neurologist may often doubt at the time of operation whether they have succeeded in laying bare the diseased area. Only by enlarging the operative field can the line of demarcation between morbid and healthy tissues be detected. Glioma may occur in any part of the brain, but it is found most frequently in the white substance of the hemisphere and upon the convexity. It is not adherent to the pia. It is essentially a tumor of slow growth, and for this reason it may cause less pronounced symptoms than the more rapidly growing tumors do. The blood-vessels in the tumor are at times so much dilated as to justify the name of telangiectatic glioma. Hemorrhages into the mass of the tumor account for the apoplectic seizures which mark the progress of the symptoms. In exceptional cases an apoplectic seizure may occur at an early stage, and the existence of the tumor may not be suspected until other symptoms set in, such as persistent headache, optic neuritis, and no tendency to recovery, which are characteristic of neoplasm and not of ordinary cerebral apoplexy.

Sarcoma may occur at any age, but is a little more frequent in adult life than tubercles and about as common as glioma. Sarcomatous growths have no special area of predilection, except that in children they are more frequent in the cerebellum than in any other part of the brain. The new growth may originate from the skull, the dura, or the brain substance itself; the connective tissue of the pia and the walls of the blood-vessels supplying the actual point of issue.

FIG. 25.—Small Spindle-celled Sarcoma, × 400. The nuclei of the spindle cells are dark colored. Capillary vessel and a few lymphosytes visible in the section.

In size sarcomata may vary much, from that of a small nut to tumors weighing many ounces. They are generally encapsulated, push the brain substance asunder, but do not infiltrate into it; whence it follows that they are enucleated more easily than other forms of tumors. In this respect they are favorable for surgical interference, but the uncertainty of their dimensions and the fact that they are often metastatic and multiple help to make the ultimate results of operation for their removal less promising. We may distinguish between hard and soft sarcomatous growths. In the latter the neoplasm has, as a rule, undergone some fatty or colloid degeneration. Myxosarcoma and cystosarcoma denote other varieties. A combination with gliomatous structure is known as gliosarcoma. An interesting

tumor of this description weighing two and one-half ounces has recently been removed by Keen for Thomas in a boy of seventeen years. If there is an excessive development of blood-vessels together with sarcomatous tissue, we speak of angiosarcoma; another mixed

FIG. 26.—Psammoma, ×50, showing glistening ball- and rod-shaped bodies.

FIG. 27.—Psammoma of the Dura Mater, ×400. *a a*, Blood-vessels; *b*, ball-shaped body, consisting of three parts; *c*, a cell with double nuclei.

form, characterized by excessive deposit of pigment and starting from the pigment cells of the pia, is known as melanosarcoma. Such growths are unusually malignant. A diffuse sarcomatosis of the brain and spinal cord has been described by a number of writers (A. Westphal and others).

Considering the frequent association of sarcomatous tissue with

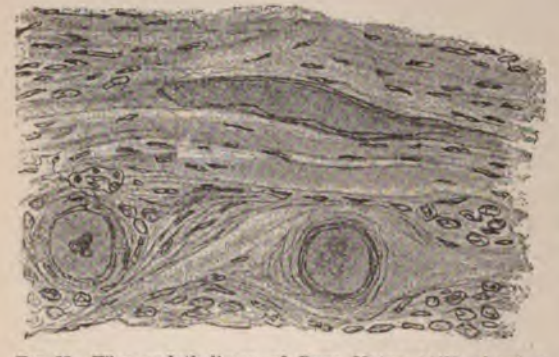

FIG. 28.—Fibro-endothelioma of Dura Mater, ×400, showing hyaline portions and round bodies with concentric arrangement of cells. The nuclei are of varying shapes.

FIG. 29.—Fibro-endothelioma of Brain, ×400, issuing from the Dura. Large cells arranged in concentric fashion and forming oval bodies.

other forms of neoplasm, it is readily inferred that the histological appearances will vary greatly; but sarcoma, pure and simple, is characterized by either round, spindle-shaped, or fusiform cells (Fig. 25). The interstitial tissue is of little note except in those

forms of fibrosarcoma which are again a mixture of fibroma and sarcoma. Endothelioma (Figs. 28, 29) is a designation for growths arising from the endothelial cells of the brain coverings.

Virchow has given the name psammoma to small sarcomatous growths filled with granular or calcareous concretions and starting from the dura mater (Figs. 26, 27). These typically *small* tumors do at times attain to a considerable size, as evidenced by the cases reported by Mitchell, Peterson, and others.

The histological characters of *fibroma* or *osteoma* need not be described in detail; both forms of tumor are more likely to occur in conjunction with other forms of new growth (Figs. 28, 29). Thus we have fibrosarcoma, osteosarcoma, etc. Osteoma starts almost invariably from the dura or from an exostosis of the skull. Lipomatous growths are very rare in the brain itself, and adenoma occurs only in conjunction with the hypophysis cerebri and is practically nothing but a proliferation of the normal tissue of this structure. The glandular tissue is also the occasional seat of carcinomatous growths.

Carcinoma has comparatively little practical value, as it is generally metastatic, and occurs in patients whose fate has been sealed by the occurrence of carcinoma in other organs. It is only worth mentioning that carcinomatous growths may vary in size from that of a small nodule to that of a large nut.

Solitary tubercles deserve more extended notice. They are generally secondary to some tuberculous deposit elsewhere in the body; but the primary seat of the trouble is often overlooked even in autopsies carefully made. In children, in whom tubercles are more frequent than any other kind of neoplasm (see Table II.), the bronchial and mesenteric glands may be the starting-point for the tuberculous invasions of the cord and brain. The size of tuberculous growths may vary from that of a pea or cherry to the very unusual size of the large tumor depicted in Fig. 30. These growths may occur singly and independently of any tuberculous meningitis; more often they constitute part of a widespread tuberculous process. Several solitary tubercles are frequently found in one and the same brain (Fig. 30). In childhood these tumors are located oftenest in the pons, cerebellum, and cortex. Starr collected 152 cases of tubercle among 300 cases of tumor of the brain in young persons; of these 152 cases 47 occurred in the cerebellum, 14 in the pons, 16 in the corpora quadrigemina and crura cerebri, 14 in the larger ganglia, 13 in the cortex, 6 in the centrum semiovale. Multiple tubercles occurred in 34 cases.

Tubercles are apt to originate in or near the pia and to grow inwards into the substance of the brain. If a tubercle have no connec-

tion with the pia it is more than probable that it started from the sheaths of the blood-vessels, where minute tubercles are so often seen in general tuberculosis of the brain and its coverings. The large solitary tubercle is supposed to be due to a union of many smaller tubercles; the marginal zone contains the round and giant cells, the central portion of the tumor contains few if any blood-vessels and sooner or

FIG. 30.—Vertical Section through Cyst *C* and two Tumors (tubercles), showing Distortion of Brain Axis and Displacement of Left Ventricle. *P*, Pons; *V*, the left ventricle.

later undergoes caseous or fatty degeneration. The solitary tubercle may grow rapidly or very slowly. It may bear a striking resemblance to a gumma, except that the latter does not often attain to the enormous proportions of some tubercles. The bacilli of tuberculosis are found in the marginal zone, and are not present with sufficient regularity to help us much in making the post-mortem diagnosis of the character of the tumor. Finally it is worth noting that a tubercle may undergo such complete degeneration as to give rise to cerebral abscess.

Gumma is seen less frequently on the post-mortem table than any other tumors of the brain. This is due to the fact that the syphilitic neoplasm is capable of absorption. Gummata and tubercles are not very unlike each other in appearance. In size there is no marked distinction; both may undergo caseous degeneration, but gumma is subject to a retrograde metamorphosis, which is not the case with a tubercle. During the retrogression fibrous tissue may be formed, and the surface of a gumma is not as a rule so smooth as that of a tubercle. But if the tumor itself is not to be distinguished from a tubercle,

there are changes in the surrounding tissue and particularly in the blood-vessels which help us to determine the character of the neoplasm. Thus it is every unusual to find gumma without distinct specific endarteritis or periarteritis of the cerebral, particularly the basilar blood-vessels, not to mention specific growths in other organs of the body.

Echinococcus cysts are so rare in this country that we can pass them by with scant notice. In European countries they are so much more frequent that German and French writers appear to be sceptical as to their non-occurrence here. Oppenheim states that in 5,300 autopsies performed in Berlin there were 87 cases of cysticercus, and of these 72 were found in the brain; in Munich there were only 2 cases in 14,000 autopsies, and in Prague cysticercus was found 28 times in 5,323 autopsies. The nature of the cyst is recognized by an examination of the fluid.

Cholesteatoma is a rare form of brain tumor; it is characterized by the presence of glistening bodies resembling mother of pearl. The name is due to the presence of cholesterin crystals. The neoplasm is supposed by Virchow and others to be of epithelial origin; this view is supported by the occasional presence of fine hairs in the substance of the tumor. An interesting case of this description was reported by Osler some years ago (1887).

Angiomata, vascular tumors, have also been observed by Bremer and Carson, Starr, McCosh, and others.

Among tumors of the brain *aneurysms* must be considered briefly, for they may cause all the symptoms of intracranial neoplasms. The male sex is a little more liable than the female. Aneurysms may occur at any age; they have been found in children seven and ten years of age. Osler has reported a similar case in a boy of six years. The Sylvian and basilar arteries are the most frequent seat of large aneurysms, but they occur also on the median cerebral artery, the internal carotid, and on all the other cerebral arteries. The aneurysm, if sufficiently large, presses upon the surrounding brain tissue. Aneurysm of the Sylvian artery will produce symptoms pointing to the involvement of the frontal and temporal lobes; the cranial nerves will be involved also. Aneurysm of the internal carotid will involve the posterior portion of the frontal lobe or the crura cerebri; it will also involve the cranial nerves (second, third, sixth, and eighth); the fourth and fifth generally escape. Aneurysm of the basilar artery will involve medulla, pons, cerebellum and cerebellar peduncles, and the cranial nerves (ninth to twelfth).

Symptoms.

Few morbid processes give rise to as distinct a series of symptoms as does intracranial tumor, and yet cases are reported every now and then in which the existence of a tumor was not suspected although the case had been observed by competent medical men. This need not be a surprise if we consider the difficulties of differential diagnosis, but it is astonishing to find that even large tumors constitute a fair proportion of the unexpected findings of the post-mortem table; for which we may give the very simple explanation that brain tissue is yielding, that it can frequently be compressed to a very marked extent without losing its function, that many of the tumors are notoriously of slow growth, that some of them do not invade the cerebral tissue but push it aside without actually destroying it, and in addition we must also consider that if a tumor grows in a silent portion of the brain it may give rise to no distinct symptoms, while the general symptoms—headache, vertigo, nausea, even optic neuritis—occur in a variety of different cerebral conditions such as meningitis, syphilitic affections of the brain, and the like. Thus some other morbid lesion and not a neoplasm may have been suspected.

In the large majority of cases, however, the symptoms are in evidence from a very early period. For purposes of classification we may divide these into two large groups: (1) General symptoms, (2) localizing or signal symptoms.

General Symptoms.

These are due chiefly to the increased intracranial pressure. Under normal conditions the brain and the cerebrospinal fluid fill the cranial cavity. Any new growth, whether it be solid or semisolid, not only increases the pressure because within a given space there is more solid substance, but also because an increase of cerebrospinal fluid is an almost invariable accompaniment of a new growth. It is in part due to this increase of cerebrospinal fluid that we have general symptoms which will appear whether the tumor be situated in the brain proper or in the cerebellum, whether it be situated upon the surface or within the structures of the brain. These general symptoms are headache, nausea and vomiting, insomnia, convulsions, and double optic neuritis.

Headache is one of the earliest and often a most striking symptom of tumor of the brain. On the whole it is more intense the nearer the tumor is to the surface of the brain, and in all probability it is the more or less direct involvement of the meninges which is the

immediate cause of the pain. There are exceptions, however, to this rule, and intense general headaches sometimes accompany neoplasms in the vicinity of the larger ganglia. In these it is to be supposed that the increased fluid, the increased intracranial pressure, explains the occurrence of intense cephalalgia. Patients describe these pains as boring or gnawing pains; not infrequently they state that the head feels as though it were ready to burst. It is to be noted that in children whose fontanelles are not completely closed, or whose skulls are so yielding that the sutures can be forced apart by increased pressure from within, the headache does not always attain to the severity which is common in adults. It is only in cerebellar tumors in children that we meet with the same intense occipital headaches as in the adult, and these must be accounted for by the unyielding character of the tentorium cerebelli. In many instances the headache is referred to a circumscribed area of the head, and if so it has distinct localizing value, for in the absence of other signal symptoms the location of a headache in a case in which there is suspicion of a tumor may be taken to indicate the approximate site of the tumor, and if sensitiveness on percussion of the skull is found over this area and over no other, it is just to infer that the tumor must be situated underneath the painful area. The suspicion of tumor is often aroused by the intensity of the pain, which is even worse than that of a trigeminal neuralgia because it is continuous or almost so; but if the pain is variable, even if it disappears entirely for hours or days or even weeks, the absence of such pain need not militate against the diagnosis of tumor. I have in mind a patient who for a period of several months had entirely recovered from headaches, yet the other symptoms of tumor persisted and on post-mortem examination a large glioma was found. Another patient, more recently under my care, presented very slight swaying for a few days, and then developed most intense headaches. These persisted for a number of weeks without any other symptom, yet the headaches were so characteristic, so intense that the diagnosis of intracranial neoplasm (probably cerebellar) was made, and this diagnosis was corroborated by the post-mortem examination, which revealed a large tumor in the cerebellum. It is highly probable that the changes in the amount of cerebrospinal fluid and the variation in the blood supply of the meninges are sufficient to explain the remissions in pain.

Sleeplessness is another important symptom. It may be the direct result of continued pain, but at times it is present even if the new growth does not cause the usual agony. It appears at times early in the course of the disease and is associated frequently with a great amount of restlessness.

The association of nausea and vomiting with headaches or insomnia should lead to the suspicion of tumor in the absence of fever and if all these symptoms persist for a considerable period of time. The vomiting is characterized by the fact that it is sudden and projectile, that it has no direct relation to the ingestion of food, occurring just as frequently before as after a meal and often following upon any change in the position of the head.

Vertigo is another common symptom of tumor and is particularly apt to occur with every change in the position of the head and in some instances whenever the person attempts to stand. In tumors of the cerebellum it is so pronounced and so aggravated that the patient reels to one side or another, sometimes forwards, sometimes backwards—a condition which is easily explained by the intimate relations existing between the cerebellum and the auditory nerve, for the latter in turn is known to hold important relations to the function of equilibrium.

Comatose and stuporous conditions have been observed with tumors of the brain, but as a rule not until the growth is well advanced or until it has given rise to a very marked increase of intracranial pressure. Before the comatose or stuporous condition is reached there is often a mental apathy which is quite characteristic of intracranial neoplasm. It is perhaps a little more common in tumors of the frontal lobe than in those occupying other sites.

Knapp believes that diminished intelligence is a regular accompaniment of cerebral tumor. While this may apply in a certain number of cases, it is very positive that many of the patients retain their full intellectual powers for a long period of time. In some instances defective speech and consequent hesitancy on the part of the patient give rise to the suspicion of intellectual defect. In exceptional cases a condition of melancholia, a maniacal state, confusional delirium, and even systematized delusions have been observed; but, after all, psychoses are comparatively rare and do not at all compare in clinical value with the other general symptoms noted above.

To the preceding general symptoms we must add also convulsions which may be general or localized. The diagnosis of intracranial tumor has generally been made before the occurrence of such convulsions, but it is well to bear in mind that the entire series of symptoms may be introduced by these, and particularly in children in whom convulsions occur very much more easily than in the adult. The convulsions may involve the entire body or may be restricted to a definite area. It is only the general convulsions that have no definite localizing value and may occur with tumor in any part of the brain, while localized convulsive seizures, as we shall see later on,

point to direct involvement of the area governing the part first convulsed. The association of convulsions with headache, with nausea, vertigo, and optic neuritis, may well give rise to the suspicion of tumor, particularly if a meningitis or an encephalitis can be excluded by the entire absence of fever. Slight febrile movements may possibly occur during the growth of a cerebral neoplasm, but the fever is never so constant as it is in the acute infectious brain diseases.

Double optic neuritis, choked disc, and complete optic atrophy are the most important general symptoms of intracranial tumor. If present there is no symptom more valuable than these; but every one should be mindful of the facts, first, that optic neuritis occurs in association with other diseases such as meningitis and general syphilitic conditions; and, secondly, that tumors may attain to a considerable size without the development of optic neuritis. The transition from an optic neuritis to a choked disc is a very gradual one. Uhthoff has suggested that the diagnosis of choked disc should be restricted to a prominence of at least two-thirds of a millimetre. The choked disc is recognized by a reddish-gray discoloration and a cloudiness of the entire disc. The veins are enlarged and the blood-vessels, in passing over the borders of the disc, cannot be followed as distinctly as in a normal papilla. If the disc is very much swollen, its limits may be entirely obscured and may seem to pass imperceptibly into the general substance of the retina. The size of the disc may be increased two- or threefold and hemorrhages may add to the complex but characteristic picture. As the disease progresses the swelling subsides and gradually passes into a condition of atrophy, but even after the atrophy has appeared some of the signs of a preceding inflammatory condition remain, and if patients are seen at a late stage of the disease it is often important to determine whether the atrophy is a primary one or whether it has followed upon a preceding choked disc or neuritis. As a rule the optic neuritis is developed equally in both eyes. If it is more developed on one side than the other, it is generally to be inferred that the neuritis is more marked in the eye corresponding to the location of the tumor; but too much dependence cannot be placed upon this symptom, or at least it has little localizing value, for a number of writers have stated that they have found optic neuritis more pronounced in the eye opposite the diseased half of the brain.

As for the frequency of optic neuritis in cases of cerebral tumors, there is a rather wide discrepancy among different writers. Gowers claims that it occurs in at least four-fifths of all cases, Knapp in two-thirds, Starr says in eighty per cent., and Oppenheim asserts that it occurred in eighty-two per cent. of his cases. Very recently I have

been able to follow two cases of brain tumor in which not a sign of optic neuritis as determined by competent ophthalmologists was present from beginning to end. It is natural to suppose that it is more constant in tumors of the cerebellum and of other basilar structures, for the effect of increased intracranial pressure would be felt most readily if the tumor were thus situated. But it is difficult to gather statistics on this point, and all that can be positively asserted is that it may be absent in tumors of the meninges, of the cortex, particularly of the occipital lobes, and even if the tumor be situated in the pons. But the character of the tumor may have an important bearing upon this fact, and since gliomatous growths occur in the cerebellum as well as in the pons, since these grow slowly and may be attended by only a slight increase in intracranial pressure, we can understand why with such growths in these structures optic neuritis may not be developed. The explanation of optic neuritis becomes all the more puzzling when we consider that relatively small tumors are at times associated with optic neuritis, and that in other cases large and even very vascular tumors in different parts of the brain do not give rise to choked disc or optic neuritis. Oppenheim states that the fundus was found to be normal most frequently in cases of cysticercus, but refers at the same time to a few observations which prove that even under such conditions the papillæ may be swollen.

Vision is commonly disturbed, but not necessarily in proportion to the amount of optic neuritis. We must suppose that a considerable swelling of the nerve is possible without interference with its function, and that the optic nerves offer a varying resistance in different individuals. In course of time the diminution in visual function becomes very marked and blindness is the unfortunate accompaniment of a large number of cases of intracranial neoplasm. It is to be noted also that blindness occurs very suddenly in some cases of tumor and that in others it is of a transitory character. Fluctuations in the amount of fluid or possibly in the condition of the blood-vessels and corresponding variations in pressure exerted upon the optic nerves must be considered in any attempt to explain this peculiar phenomenon. Bruns claims that transitory amblyopia occurs more frequently with tumors of the occipital lobe than with those in any other position.

Sudden variations in the pulse rate, rapidity and irregularities of respiration have been observed. In children I have seen Cheyne-Stokes respiration in cases of cerebellar tumors long before the terminal stage had been reached.

Localizing or Focal Symptoms.

While the existence of tumor may be suspected from the presence of the general symptoms, its exact location cannot be determined unless we consider the special, localizing symptoms. These may be subdivided again into direct and indirect symptoms. The direct are those which are caused by the immediate action of the tumor upon an area that has distinct functions, while the indirect symptoms are exerted by a tumor situated at a distance and probably through the effect of the increased pressure or by actual displacement. Thus in one of my cases a large tubercle had so distorted the entire brain axis that almost every nerve at the base of the brain was affected (Fig. 33). The indirect symptoms are on the whole more frequent in cases of cerebellar tumor than in any other. Thus, in this class of cases we have the general symptoms of intracranial neoplasm together with reeling and distinct occipital headaches; associated with these is a paralysis of the rectus externus supplied by the sixth nerve which is almost pathognomonic of cerebellar tumor. This sixth-nerve paralysis is an indirect symptom of cerebellar tumor and is caused by pressure upon the nerve lying between the base of the brain and the skull, such pressure being due to the pushing forwards of the tentorium, compelling an impingement of the pons and medulla upon the base of the skull.

The focal symptoms can be inferred from the study of the functions of the brain as given in a previous article. The various cortical areas have been determined, as is well known, by experiments upon animals (dogs and monkeys chiefly). In man the effects of disease have shown that in the human brain the localization of function is quite as accurate as in animals, and this knowledge has been gained largely by the clinical and pathological study of tumors of the brain. Electrical tests performed in the course of operations upon the exposed dura and cortex have proved to me that there is the same minute subdivision of function in the cortex of man as in that of the lower animals. Assuming that the reader is familiar with the general facts regarding localization of brain functions (see p. 33), it will not be necessary to review them in detail, yet it will be well to recite the chief symptoms as observed in tumors according to the variation in location.

Tumors of the Cortex.—It is of the utmost importance to determine whether or not a tumor is situated in the cortex, for such a neoplasm may be within reach of the surgeon's knife, whereas those far removed from the surface are not at the present time proper subjects for surgical interference. It is not an easy matter, however, to distin-

guish between the purely cortical tumors and those situated in the subjacent white matter. Some claim that it is impossible to differentiate between cortical and subcortical tumors; but, as I have said

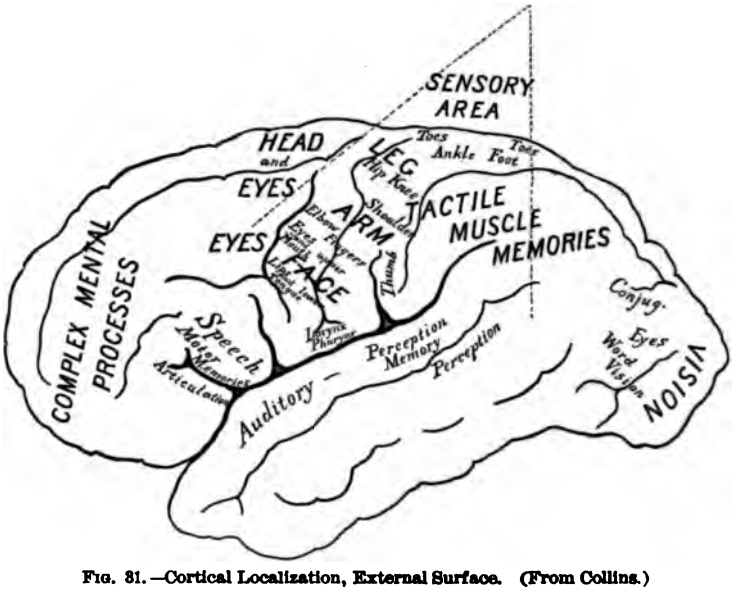

FIG. 31.—Cortical Localization, External Surface. (From Collins.)

in a previous publication, this would seem to be overstating the facts. While the localizing symptoms may be very much the same in both these classes of tumors, the order of development of the symptoms

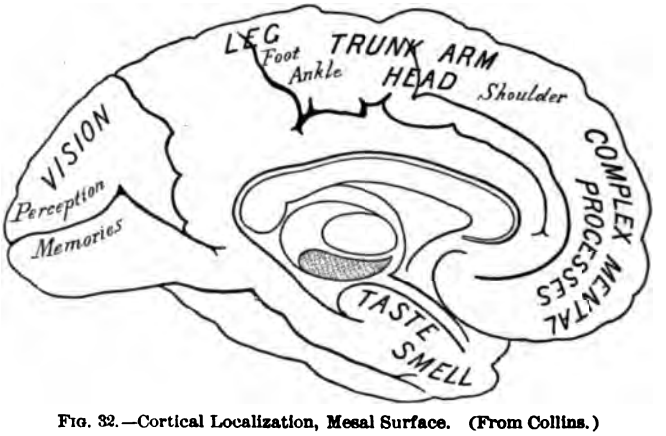

FIG. 32.—Cortical Localization, Mesal Surface. (From Collins.)

will be different and may give some indication of the exact site of the tumor. Take, for instance, tumors in the motor area: those in or near the gray matter, however small, will give rise to occasional con-

vulsive seizures at the very beginning of the disease, whereas the tumors which begin in the subjacent white matter and gradually extend into the cortex proper will cause paralytic symptoms long before they give rise to symptoms of cortical irritation. Interference with the functions of the fibres radiating downwards from the cortex will cause paralysis, but the convulsive seizure is purely a matter of irritation of the cortical cells proper. If the headaches have increased *pari passu* with the development of irritation symptoms, the inference that the tumor has grown from beneath the cortex into the latter is a very just one. It is also to be borne in mind that certain classes of tumor are more apt to begin in the cortical tissue than in the subcortical white substance. Thus tubercles, gliomata, and above all gummata are almost certain to begin near the surface, while sarcomata and cysts are as often subcortical as cortical in origin.

Tumors of the Frontal Lobe.—The frontal lobe has for a long time been considered to be one of the silent areas of the brain, and tumors of this region have occurred without giving rise to any distinct localizing symptoms, and frequently it was only the persistence of pain in the frontal region that aroused the suspicion of a tumor in the anterior portion of the brain. The uncertainty of this diagnostic symptom was increased by the fact that frontal headaches have been observed even with occipital tumors. If the tumor extends downwards and involves the olfactory bulb the sense of smell may be diminished or lost; but during late years, and particularly in consequence of Flechsig's ingenious theories and his array of facts, we have learned to know that the frontal lobe has important relations to the higher psychic functions of man. That such impairment of psychic functions would occur was made altogether probable by the facts published many years ago by Goltz, who discovered a very marked change in the behavior of his dogs after he had excised or destroyed the frontal lobes. Bernhardt, basing his statements upon statistics gathered a number of years ago, was inclined to deny the existence of these symptoms in man, but the more careful observations of recent years have shown that while psychic changes may occur in conjunction with tumors in almost any part of the brain, they are more common in tumor of the frontal lobes than in those affecting other areas. In a case operated on by McBurney, Starr was guided by the existence of mental symptoms in indicating the site for operation; but the fact that this proved successful in a single instance does not warrant us in attempting to use the symptom in this way unless the other features of the case lend support to such a focal diagnosis. Oppenheim, who summed up this entire subject very carefully, is of the opinion that in those cases in which mental defect, apathy, stupor,

or some well-defined psychosis has constituted a striking symptom of the disease the suspicion of a frontal tumor may be entertained, but he does not believe that we are justified as yet in accepting this as a safe guide in focal diagnosis.

The situation of the tumor in the frontal lobes can be inferred with much more certainty if in the course of its growth it gives rise to symptoms which indicate its proximity to the motor areas, but before it transcends the limits of the frontal lobe it is very apt to cause speech disturbances which are characteristic of lesions of the third frontal convolution in the left hemisphere. Pure motor aphasia—a form of speech disturbance in which the patient is perfectly able to understand everything that is said to him but cannot find the words to express his thoughts—is the signal symptom of a morbid process affecting this area. Thus in a case which I had occasion to observe within the past year, a young woman, the sister of a physician, had become depressed and apathetic. This mental condition existed for a number of weeks so that her brother, the physician, concluded that she must have a change of surroundings to improve her mental condition. It was only after the lapse of weeks that she developed a hesitancy in speech, and this consisted at first in the loss of proper names so that she would paraphrase in speaking, not being able to find the word to suit the object or the person she wished to designate. After a further lapse of weeks speech became more and more defective so that she could use only a very limited number of words. Evidently in this case the tumor had started in the anterior portion of the frontal lobe, had gradually invaded the third frontal, and since the sensory part of speech also became involved we assumed that the tumor had directly or indirectly affected the first temporal convolution. In this case there was no optic neuritis but a slight weakness of the right upper extremity, and a slight increase of the reflex in the same proved that there was a morbid process present which was gradually involving the adjacent arm area. An operation was proposed and a large gliosarcoma was found in the upper portion of the third frontal convolution; it was of considerable size and had gradually destroyed the function of the diseased parts. Aphasia cannot, however, be considered as indicating a morbid process in the third frontal convolution only, for, in view of the intimate relations existing between the motor and other speech centres, disturbances in language may occur, and in order that a correct diagnosis should be arrived at it is important to analyze the exact form of aphasia present in a given case (for particulars the reader is referred to the article on Aphasia).

The proximity of the first temporal convolution to the third frontal will explain why sensory aphasia may be added to motor aphasia

in a number of instances. There may be a doubt even as to the location of the tumor in the left or right hemisphere. In left-handed persons the left hemisphere is entrusted with the functions of speech, but it is a matter of dispute whether the right hemisphere is so completely devoid of all speech function that slight speech disturbances may not be due to a tumor in the third frontal convolution of the right half. While great care should be exercised in utilizing the symptom of aphasia, particularly in operative cases, the association of pain on percussion over a region corresponding to the left third frontal convolution or the occurrence of spontaneous pain in this circumscribed area may be considered corroborative evidence of the highest value. If the aphasia is associated with symptoms, however slight, in the right half of the face or in the right upper extremity, the probability of the location of the tumor in the left hemisphere is very great. The association of agraphia with motor aphasia is very common and was most distinct in the patient referred to above, who had not only forgotten to write her name, but could not even copy it if the name was written and shown to her. Some years ago I called attention to the fact that in young children the differentiation between the right and left hemispheres in the matter of speech is not nearly so complete as in the adult; for that reason the occurrence or non-occurrence of aphasia in children suspected of intracranial neoplasm must be used with the greatest caution.

Automatic and forced movements have been observed with tumors of the frontal lobe, but they have no diagnostic value as localizing symptoms, for they occur with tumors in other parts of the brain and particularly with those involving the larger internal ganglia.

Tumors of the Motor Area.—Tumors in this area are recognized most readily because they give rise to a distinct set of focal symptoms. The experiments on animals, particularly those of Munk, Ferrier, Beevor, Horsley, and others, have shown that in animals the motor area is included within the anterior and posterior central convolutions. In man the anterior central is more distinctly involved than the posterior central, but it is probable that both these convolutions belong to this area, although the anterior central has become the more highly specialized part. The exact division of these areas has been alluded to in a previous article and it will suffice for our purposes to say that, beginning with a portion of the anterior central convolution nearest the fissure of Sylvius, we have the part representing the movements of the tongue and face; above this the movements of the thumb, the fingers, the hand, the arm, the shoulders, and the leg, in the order just mentioned. Roughly speaking, the movements of the legs are governed by the upper third of the

motor area, the movements of the upper extremity by the middle third, those of the face and tongue by the last third. In the last named there are also centres governing the throat, gullet, and jaws; in the middle area there is a specialized thumb centre, and in the leg area the hips and knees, the foot and toes, and the large toe have specialized centres (see Figs. 31 and 32). It is well to remember that these centres do not govern muscles so much as they govern movements. Using electrical tests, we can succeed in eliciting definite movements (for instance, of the angle of the mouth) by applying the electrodes to definite and well-circumscribed small areas. Each motor area represents not only the movements governing motion in the opposite half of the body, but has also certain relations to the same half of the body. This may account in part for the fact that on removal of a definite portion of the motor area complete paralysis of the part governed by it does not necessarily follow, or at least is not permanently maintained. This has been observed by me again and again in cases both in children and in adults after the excision of a special centre.

There has been much discussion for many years as to sensory representation in these motor areas. While I do not believe that sensation is exclusively represented in these centres, the facts point towards a partial representation of sensation in the motor areas or in parts immediately adjoining them. This is made evident by the fact that after excision of a given centre, say the arm centre, numbness and paræsthesiæ accompany paralysis of the part governed by the excised area; but sensation seems to be restored even more quickly than motion, and for this reason it is fair to assume that the motor centres are not the only parts of the brain in which sensation is represented. On the other hand, the mere fact that sensation is so frequently affected after excision of parts of the motor area proves beyond reasonable doubt that the so-called motor centres are in part sensory in function. Cases have been reported by Edinger and others in which a definitely localized pain has been the most prominent symptom following upon tumor in the motor area.

The most characteristic symptom of tumor in any part of the motor area is cortical epilepsy. By this we mean convulsive seizures affecting the part governed by the diseased area and coming on with or without loss of consciousness. The epileptic discharges are developed in a definite order; the part first convulsed indicates the special area in which the disease is most marked; from this part the irritation spreads to neighboring areas, and thus the convulsive movements may gradually affect all the members of the body. If, for instance, the arm centre is the site of a tumor, twitching movements in

the fingers or the wrist will be first noticed; after this the convulsive movement may spread either to the face or to the leg, and by degrees may involve the opposite half of the body. While consciousness may be preserved during the entire attack, it is lost in the majority of cases as soon as the convulsive movements are established in both halves of the body. Such epileptic seizures may be frequently repeated. At times they are so slight that they are interpreted to be nothing more than slight twitching movements and are often overlooked by the attending physician; but the regularity and the rhythmical character of slight clonic movements in any definite part of the body should arouse the suspicion of organic disease as the cause. The epileptic seizures may be repeated at shorter or longer intervals; I have known as many as forty and fifty seizures per day, and other cases in which a seizure, particularly in the beginning of the disease, would occur only every few days. Remissions occur even if the tumor not only persists but grows steadily. Thus in one patient there was a complete remission of three months, and I was led to believe that the morbid process had subsided, when suddenly an apoplectic seizure occurred in which the patient died, and on the post-mortem table we found a large glioma which had evidently been growing steadily for months and months. The convulsive seizures are most apt to be established in the earlier period of the disease during which the tumor acts as an irritant upon the cortical gray substance. As soon as this substance has been destroyed by the invasion of the new growth the irritation symptoms subside, that is, the convulsive seizures cease; but the paralytic symptoms, denoting complete destruction of cortical substance, supervene. The paralytic symptoms are very likely to persist in the form of a monoplegia or it may be even more limited in extent. Thus Macewen and Keen have reported cases in which the extensors of the big toe or the muscles of the thumb were the only parts paralyzed for a considerable period of time. It is on the strength of these strictly localized symptoms, paralysis and numbness of limited parts of the body, that we can recognize the exact site of a tumor, but of course we have no right to diagnosticate tumor unless the general symptoms are present in addition to the special localizing signs. Furthermore, it is worthy of note that partial epilepsies have a definite localizing value only if they appear early in the course of the disease. If general symptoms of tumor of the brain have existed for a long period and then a partial or Jacksonian epilepsy is developed, great care must be exercised in definitely locating the tumor, for it is more than probable that the tumor has invaded the motor areas secondarily and its beginning may have been in a distant part of the brain. The exact character of the

motor symptoms will be best understood by reference to Figs. 31, 32, denoting the distribution of the motor areas. To complete the symptoms of tumor of the motor area we need merely add that ataxia has been observed in some cases; vasomotor and secretory disturbances in others. Salivation has been noted by Oppenheim and Koehler, but this same phenomenon has been referred to by Torjé in tumors of the frontal lobe, so that it cannot be credited with any special value. Tachycardia has also been observed, as have been disturbances of the function of the bladder.

Tumors of the parietal region, in which are to be included the superior and inferior parietal lobules, have a special importance, since they may give rise to distinct localizing symptoms in consequence of their proximity to the anterior and posterior central convolutions. Such tumors may give rise to changes which have been noted in connection with involvement of the motor area; but, according to the observations of Dana, Starr, and others, the parietal tumors are more apt to cause sensory than motor symptoms; disturbances of tactile and of muscular sense have been observed. It is questionable whether these tumors cause such symptoms by involvement of the posterior central convolution or by disease of the sensory fibres passing underneath this portion of the cortex to the central region of the brain. In a case published by Starr and McCosh there was good evidence presented of the existence of a distinct centre for muscular sense, but further evidence is needed on this point before we can claim that muscular sense is represented exclusively in this part of the brain and in no other.

Tumors in the border line between the parietal and occipital lobes may give rise to bilateral homonymous hemianopsia by involvement of the white tract of Gratiolet, which passes from the internal capsule to the occipital lobe. Ferrier's claim, that the centre of vision lies in the angular gyrus, is explained by the involvement by disease in this region of the optic radiations just mentioned. By extension to the left inferior parietal lobule the tumor may give rise to that special disturbance of speech known as word blindness; the patient cannot read and write at will, though he may be able to write upon dictation or to copy written signs. According to Wernicke the conjugate movements of the eyes are governed by a centre in the inferior parietal lobule. If there is distinct impairment of this one function, disease of this region may be suspected.

Tumors of the Occipital Lobe.—New growths in this region are recognized by those disturbances of vision which we include under the term hemianopsia: loss of vision in one-half of the retinal field. As each occipital lobe is connected with one-half of each eye, tumor

in this region is characterized by bilateral homonymous hemianopsia without any other special symptoms. In point of differential diagnosis it is well to remember that hemianopsia may be due to lesions in the optic tract or optic radiations as well as in the occipital lobes; but if the disease is situated in the former areas it is apt to be associated with motor, sensory, and pupillary symptoms. The researches of Nothnagel and Seguin, which have been corroborated by others, point with a great degree of certainty to the cuneus as the actual centre of vision. We must therefore consider the possibility, in a case in which hemianopsia is the chief symptom, of a tumor beginning on the median surface of the occipital lobe and gradually extending to the outer surface. The later investigations of Henschen prove, however, that a lesion in almost any part of the occipital lobe, particularly if it is sufficiently large to compress the white matter underneath the occipital cortex, may produce hemianopsia, so that any part of the occipital lobe may be the starting-point of a tumor giving rise to hemianopsia. It is in these cases of tumor of the occipital lobe that we also meet with that peculiar condition in which the patient exhibits what is known as psychic blindness, seeing objects but no longer recognizing them or the uses to which they are put. This is more apparent in lesions of the left side in right-handed persons, and of the right half in left-handed persons.

Tumors of the Temporosphenoidal Lobe.—This portion of the hemispheres contains the centres for hearing and for sensory speech; it is therefore natural to expect that in a case of tumor in these auditory regions hearing will be impaired but not abolished on the side opposite the lesion, and sensory aphasia will be present. The patient is able to speak spontaneously and correctly; is not able, however, to understand what is said to him, and of course not able to repeat spoken language.

Distinct as are the symptoms of lesion in the temporal lobes, a tumor in this region, by crowding neighboring parts of the brain, is very apt to produce motor aphasia as well as paralysis of the face, tongue, and lips. The temporal lobe also contains the association tracts between the motor-speech centre and the visual centres. A tumor in this region is therefore apt to give rise to complicated speech disturbances, such as alexia and agraphia, showing that the concerted action of these centres has been disturbed. These sensory speech disturbances are more frequently associated with tumor in the left temporal convolution than in the right, but cases in which the same symptoms have been produced by disease of the right temporal lobe have also been recorded. Symptoms denoting irritation of the acoustic nerve may introduce a series of phenomena following upon

tumor in this region; tinnitus and acoustic paræsthesiæ of all sorts are included in this category. I have not observed this in tumor of this region, but have noted it in cases of abscess. The differential diagnosis between tumor and abscess applies particularly to growths in this region, for on account of the proximity of the temporal convolutions to the bony structures of the ear abscess is developed more frequently in this region than in any other.

Bristowe and others have attempted to make out a distinct set of focal symptoms for tumor of the corpus callosum, but these are of a negative character chiefly, viz.: slight development of general symptoms (headache, optic neuritis, etc.), slight paresis of the opposite half of the body, dysarthria rather than aphasia. The occurrence of imbecility is at least one positive symptom.

Tumors of the Basal Ganglia.—Neoplasm involving the basal ganglia and adjacent parts is not rare. The symptoms produced are for the most part due to direct or indirect involvement of the internal capsule. As this capsule contains the entire motor tract for the opposite side of the body, the sensory fibres, the fibres of special sense, the speech tracts, it is easy to see that an unusual variety of symptoms may result from such a lesion. The position of the fibres in the internal capsule has been well established and in the case of very small tumors the order in which the different functions are lost may give some clew as to the direction in which the tumor is extending. The tumor may also impinge upon the lateral ventricles and thus cause considerable disturbance. As all the structures are crowded into relatively small space in this part of the brain, small tumors may cause a large variety of symptoms, and in view of the distortion produced by them we may have symptoms resulting from pressure upon the cranial nerves coming off from the brain axis below this region. The differential diagnosis between tumors of the ganglia and tumors of the cortex is based in part upon the absence of repeated convulsive seizures in the case of ganglionic tumors, and upon the development of a large series of special symptoms with relatively few general symptoms, thus showing that a small neoplasm has given rise to a greater series of symptoms than small tumors would be able to produce if located in any part of the cortex. For a long time hemichorea and hemiathetosis were supposed to be almost pathognomonic of thalamus lesions, but it is doubtful whether they have any special localizing value, since these same disturbances of motion have been observed in conjunction with cortical disease; and it is altogether probable that if they do occur in connection with disease of the thalamus, it is the irritating effect of the latter upon the neighboring pyramidal tract that is the immediate cause of these symptoms. If,

however, the general symptoms point to a location in the interior of the brain and not in the cortex, the presence of hemichorea or hemiathetosis is sufficient to warrant the diagnosis of a lesion in or near the thalamus.

Tremor and forced movements have also been observed in connection with tumors of the ganglia, but these do occur too with neoplasms in other parts of the brain. Many years ago Nothnagel insisted that a focal disease of the optic thalamus produced a mimetic palsy of the opposite half of the face. This implies that there is normal innervation of the facial muscles in voluntary movements, but that in special acts, as of laughing, the affected side does not take part. This observation of Nothnagel has been corroborated so frequently that we cannot deny it a certain diagnostic value.

Pain and paræsthesia in one half of the body have been noted in connection with tumors of the thalamus, but there may be entire absence of all sensory disturbance in cases of thalamus tumor.

Amblyopia and amaurosis have been noted in a number of cases of tumor of the optic thalamus, but inasmuch as these were generally combined with optic neuritis or choked disc it is doubtful whether any special significance can be attached to these symptoms, and as it is doubtful even to the present day whether the optic thalamus has any definite relations to vision except possibly as an agent in the conduction of reflexes, all facts relating to this subject must be accepted with great reserve. Dercum has found Wernicke's symptom (hemianopsic pupillary immobility) in a case of this description.

Tumors of the Crus Cerebri.—A neoplasm in this region is to be recognized by the association of oculomotor symptoms with paralysis of motion and of sensation in the opposite half of the body. The eye will exhibit ptosis, paralysis of all the external muscles except the rectus externus and the superior oblique, and complete paralysis of the sphincter iridis and the ciliary muscle. Both peduncles are so close to one another that a tumor occurring in one may involve the opposite side, at least press against it with such force as to interfere with its functions; for this reason it is not uncommon to find paralysis of both halves of the body, or possibly ptosis and double oculomotor symptoms with tumor in this region. For a time the symptoms may be unilateral, and as the growth increases they become bilateral.

Tumors of the Corpora Quadrigemina.—The corpora quadigemina are in such close proximity to the peduncles that tumor in the former will be very apt to be associated with the symptoms characteristic of tumor in the other, and yet there are a few distinct symptoms which point to the corpora quadrigemina as the special seat of the lesion.

This is due particularly to the relation which the corpora quadrigemina bear to visual function and to the connection between the former and the cerebellum. Lost of pupillary reflexes, nystagmus, vertigo, and a condition resembling cerebellar ataxia point to the region of the corpora quadrigemina as the special site of the tumor. All the symptoms were well illustrated by a case of a child which was three years of age, whose history I have previously published. The anatomical conditions in this case are represented in Fig. 33. This child at first presented a double ptosis, but no other ocular paralysis. It would sit quiet all day long, but if it attempted to walk it would stagger and fall. There was no anæsthesia or ataxia, and the knee jerks were absent. There was ptosis of both eyelids, the pupils being half covered, but no other paralysis of the ocular muscles and no nystagmus. There was at first merely a suspicion of optic neuritis. A month later double and complete ptosis was developed. There was no upward or downward movement of either eye; both internal recti muscles were thrown into a condition of clonic spasm when an attempt was made to use them. There was some reflex contraction during accommodation, and also slight contractility to light in both pupils. A slight paralysis of the left half of the face was noted, as was also decided impairment of vision. The child was in a condition of semi-stupor but was able to walk a little, and in so doing exhibited a most characteristic cerebellar gait with a tendency to fall to the right side. The reflexes were exaggerated in the upper as well as in the lower extremities, and there were distinct occipital headaches. The child grew rapidly worse, had convulsive seizures, became blind, comatose, and two weeks before death developed left hemiplegia. Death occurred four months after the onset of the symptoms. A solitary tubercle was found in the right lateral sinus and other tubercles occurred in the cerebellum, but the tumor found in the region of the corpora quad-

FIG. 33.—Section Passing through the Posterior Quadrigeminal Bodies. *T*, Solitary tubercle; *P. Q*, posterior quadrigeminal body.

rigemina was the one which gave rise to the majority of the symptoms. It occupied the tegmental portion of the crus, and almost completely occluded the aqueduct of Sylvius.

The more recent studies of Nothnagel, Christ, and others have been corroborated by this case, and in summarizing we may say that it is just to suspect tumor in the vicinity of the corpora quadrigemina if there is slow onset of oculomotor symptoms with cerebellar ataxia, with vomiting and optic neuritis.

Tumors of the pineal gland give rise to very much the same symptoms as those of the corpora quadrigemina, though it is supposed that the oculomotor symptoms are not quite so prominent, but that the symptoms to be referred to the fourth and sixth nerves prevail. Nystagmus, associated particularly with movement of the eyes upwards, is said to be characteristic of tumor in this region.

Tumors of the Pons and Medulla.—Tumors in this region give rise to a multiplicity of symptoms. This is explained by the large number of tracts passing through these parts, by their close proximity to each other, and by the nuclei of the many cranial nerves situated in this part of the brain. While motor paralysis may be unilateral, it is often bilateral as a result of tumor of the pons and medulla. In addition to hemiplegia and double hemiplegia we may have other symptoms pointing to an involvement of the various cranial nerves. A neoplasm in the upper half of the pons may give rise to a hemiplegia of one side of the body, with involvement of the third and fifth nerves of the opposite side. Its proximity to the crus cerebri will account for involvement of the third nerve; if the tumor is in the lower half of the pons, the fifth, sixth, seventh, and eighth nerves will be more or less involved, and the symptoms resulting from this affection will be paralysis of the rectus internus, paralysis of all the branches of the seventh nerve in one half of the face, and loss of hearing in one ear. These cranial-nerve symptoms will be on the side of the lesion and opposite to the hemiplegia. If the sixth-nerve nucleus is affected, there will be in addition distinct paralysis of one rectus externus muscle, and paralysis of the conjugate movements of the eyes towards the side of the lesion, for this nucleus is connected with the third-nerve nucleus of the opposite side and governs the outward movement of each eye. In spite of this conjugate paralysis, each internal rectus may exhibit normal movements if examined separately. If the lesion is near the surface and away from the nucleus it will involve the root of the sixth nerve and will cause paralysis of the rectus externus muscle of one side, but it will not affect the conjugate movements of the opposite side. If the patient is asked to look towards the side of the paralyzed rectus, or towards the side of the tumor, the

opposite eye will move promptly, the affected eye remaining fixed. This differentiation between the isolated paralysis of the rectus externus and paralysis of the conjugate movements is the most valuable and perhaps the only differential point of diagnosis that helps at times to distinguish between a tumor near the surface and one within the substance of the pons. The difficulties of diagnosis are increased by the great variability in the symptoms, and by the fact that some nerves in the pons escape, while others may be intensely affected.

If the tumor is situated in the medulla oblongata the symptoms will remind one of bulbar palsy. They will point to involvement of the glossopharyngeal, of the vagus, of the spinal accessory and of the hypoglossal nerves, and there will be a unilateral or bilateral paralysis of the arms and legs. The facial nerve will remain exempt. Difficulties in deglutition, in respiratory and cardiac movements, paralysis or spasm of the sternocleidomastoid and of the abducens, paralysis and atrophy of the tongue, together with vomiting, with glycosuria or polyuria, all these will suggest a lesion in the medulla. Tumors in this region, particularly gummata and diffuse gliomata, are not rare. Difficulties in diagnosis often arise on account of the bilateral character of all the symptoms, but this can be easily accounted for by the fact that the two halves of the brain at this level are scarcely separated from one another, and that it is natural for all the vascular lesions to produce bilateral symptoms. Since the sensory tracts are in close relations with the motor in this part of the brain axis, the symptoms will be still further complicated by the occurrence of partial or complete or double hemianæsthesia. The proximity to the cerebellum also opens up the possibility of another distinct series of symptoms in the case of tumor in the pons and medulla. Optic neuritis is developed as a rule early in the course of the disease and occipital headaches are particularly severe.

Tumors of the Cerebellum.—New growths in the cerebellum are not only frequent, but they also give rise to a most distinct set of symptoms. These symptoms are in part due to interference with the functions of the cerebellum itself, and to an even greater extent to the effect which the tumor produces by pressure upon neighboring parts. The majority of these growths, among which tubercles and gliomata are the most common, occur in either cerebellar hemisphere or in the vermis. The signal symptoms of cerebellar tumor are supposed to be due to interference with the special cerebellar function, namely, the maintenance of equilibrium, but it is still a matter of doubt whether this function resides in the hemispheres or in the middle lobe. There is much in favor of the view that the middle lobe bears a more important relation to this function, for in various statistics

that have been gathered on this point incoördination was found to be present in about eighty per cent. of the cases if the tumor was in the vermis and only in forty-nine per cent. if it was in the hemispheres. Yet it will be seen from these very figures that the opinion which has been advanced, particularly by Starr, that cerebellar ataxia points to involvement of the vermis or to a tumor in the hemispheres near to and compressing the middle lobe, is too positive; but as he also states that the same symptoms may be produced by tumors which compress the peduncles of the cerebellum, especially the middle peduncles in

FIG. 34.—Gliosarcoma of the Cerebellum. Early appearances of facial and auditory nerve symptoms. Case observed by Stieglitz and the author.

their course to the pons or the superior peduncles in their way to the corpora quadrigemina, he includes a sufficient territory to bring almost all cerebellar tumors under this head. In a case of sarcoma of the cerebellum which was situated laterally (Fig. 34), which I had occasion to observe to the time of the post-mortem examination, staggering was one of the earliest symptoms, and yet the tumor was considerably removed from the middle lobe. As a matter of fact, however, that form of incoördination which we describe as cerebellar ataxia, or reeling gait, or cerebellar titubation, is by far the most characteristic symptom of cerebellar neoplasm. This ataxia is characterized by a tendency to reel or to fall to one side. Whether this is always, as some claim, to the side opposite the tumor is questionable, and patients not only fall to one side, but sometimes have a distinct tendency to fall forwards or backwards; the direction in which the patient is in the habit of falling, however, generally remains the same throughout the course of the disease. As the disease progresses the

reeling becomes so marked that the patient is not able to stand even for an instant unsupported, and in extreme cases while lying in bed is not able to raise the head without great discomfort.

In addition to cerebellar ataxia we find often a marked tremor bearing a superficial resemblance to the tremor of multiple sclerosis and yet having a more atactic character than the ordinary tremor of multiple sclerosis. Strümpell has recently claimed that the tremor of multiple sclerosis is closely akin to an ataxia. While this may not be strictly true of multiple sclerosis, it is true beyond a doubt of the disturbances of motion observed in these cerebellar neoplasms. Paralysis is another common symptom in cerebellar tumors; it is evidently due to compression of the pyramidal tracts in the pons and medulla. As these tracts closely approximate each other, we can readily understand why in some cases we have a hemiplegia often of the opposite side of the body and at other times a more or less complete paraplegia, both of the upper and of the lower extremities. The face generally escapes unless the tumor is situated laterally and presses directly upon the facial nerve. In some instances even before facial paralysis is developed the involvement of the facial nerve can be diagnosticated, as it was in the case represented in Fig. 34, by the reaction of degeneration existing long before a marked paralysis had been developed. The paralysis in all these cases is of slow onset; it reminds one rather of a general weakness of the system than of the paralysis due to destruction of some part of the motor tract. In a young child whose condition I was allowed to see at varying periods the state was one which we could properly describe as a myasthenia for fully five to six months. There was no other accompanying symptom except a subnormal condition of the reflexes, and until the ataxia appeared later in the disease the diagnosis of any neoplasm and particularly of a cerebellar neoplasm could not be made. Months after the general weakness of the muscular system had existed the other symptoms of cerebellar neoplasm were developed and the autopsy proved that there was a large sarcoma in the middle lobe of the cerebellum.

There are other symptoms which deserve notice in a description of cerebellar tumor, but these are distinctly due to compression of neighboring parts. As this factor is a very varying one, the symptoms in question may be present in some and absent in many others. The most important of these symptoms is some form of paralysis due to direct pressure upon the cranial nerves. According to the amount of pressure exerted we may have a partial or even complete involvement of the oculomotor nerve, paralysis of the motor branch of the trigeminal, and pain in one half of the face indicating involvement of

the sensory branches. The fourth nerve may also be affected, but isolated paralysis of this nerve is extremely rare in this as in other conditions. Paralysis of the external recti muscles is unusually common, and is to be explained by the fact that the thin sixth nerves supplying them lie between the pons and the skull, and are most easily squeezed by increased pressure. I have seen a number of cerebellar neoplasms in which there were present the general symptoms of tumor, cerebellar ataxia, and paralysis of the recti externi, and these symptoms only.

To the paralysis of the seventh nerve we have alluded above. It is present in the fewest cases only, and if present is of the greatest importance, for it gives an indication of the probable site of the tumor in the lateral half of the cerebellum. The same may be said of auditory-nerve symptoms which occur in only a relatively small proportion of the cases. Defective hearing on one side and abnormal electrical reaction of the auditory nerve will help to determine the site and to corroborate the presence of neoplasm. Oppenheim refers to the fact that spasms may occur in cerebellar neoplasm, but is also of the opinion that these may be of reflex origin, and not due to direct pressure upon the nerve supplying such muscles. According to the direction in which pressure is exerted, the nerves issuing from the crus and pons or those having their nuclei in the medulla may be affected. Thus we may in some instances have no oculomotor, abducens, facial, or auditory symptoms, but may have interference with cardiac and respiratory action or even a glossoplegia from interference with the nuclei of the ninth, tenth, and twelfth nerves. Singultus is a not infrequent and often very troublesome symptom. Gaping I have noted, particularly in cerebellar tumors of children, and salivation is common; but I repeat what was said in a previous portion of this article, that salivation occurs with tumors in different parts of the brain and it cannot therefore be said to have any localizing value. Rigidity of the neck is often present and is a symptom which may give rise to mistakes in diagnosis, as it is present in so many other conditions, in meningitis, caries of the spine, etc.

The behavior of the reflexes has given rise to considerable discussion. In many of the cases they are increased, in others they are distinctly subnormal or even absent. Some authors are inclined to believe that if they are absent they denote a degeneration in the posterior columns as a complication of cerebellar neoplasm. While cases of cerebellar neoplasm associated with tabes have been reported, it cannot be denied that the abolition of the reflexes is so frequent in cerebellar tumors that it must be supposed to be a symptom due to the cerebellum itself and not necessarily to a complicating degenera-

tion of the posterior columns. Dinkler is willing to account for the degeneration of the posterior columns that has been found in such cases on the supposition that this degeneration is due to toxic products caused by the tumor. But it will not be necessary to resort to any such theory until it is proved by a sufficiently large number of cases that such degeneration is present in cases of cerebellar neoplasm. My own impression is that the diminution or abolition of the reflexes is much more common in cerebellar neoplasm than is the reverse state. Anosmia has been reported in some cases and nystagmus is quite frequent, but both these are evidently indirect symptoms.

The preceding account will give a sufficient idea of the large number of special symptoms present in cases of cerebellar tumor. In addition it may be observed that the general symptoms of intracranial neoplasm are on the whole more marked in cases of cerebellar tumor than in most other new growths within the skull. This can be accounted for by the fact that most of these cerebellar neoplasms are accompanied by a hydrocephalus of extreme degree. Vomiting and headaches are rarely absent in these cases. The headaches are generally occipital, but sometimes frontal. Optic neuritis is almost invariably present, and its greater frequency may be accounted for by the greater implication of all basilar structures in cases of cerebellar growths.

If we review the special and general symptoms of these growths it would seem impossible to mistake the position of a tumor if it be in the cerebellum, but it is only fair to add that the diagnosis sometimes has to be made by exclusion, and that at times cerebellar tumors exist without giving rise to any special set of symptoms and are one of the many surprises of the post-mortem table. The intimate relation existing between the cerebellum and the corpora quadrigemina on the one hand and the auditory nerve on the other is calculated to lead to occasional mistakes in diagnosis. Thus I recall distinctly a young boy whom I saw many years ago, who presented marked cerebellar ataxia which was so troublesome that the boy could only get about by walking by the wall of the room, not venturing into the middle of it. All the symptoms in this case persisted for six days. The external examination of the ear was negative and yet quite unexpectedly all the symptoms disappeared after the opening of an ear abscess which was followed by the boy's complete recovery. It is well to express a caution that the comparative ease in diagnosticating cerebellar tumor should not mislead the physician, and that however positive the symptoms may be, a thorough examination of the ear should be made before such a grave diagnosis is reached.

Before concluding the discussion of the symptoms of intracranial

cording to the varying site of the lesion we must make a brief to the tumors issuing from the base of the skull. These may mata or carcinomata, but almost all other forms may be found y produce similar symptoms if they issue from the meninges the basilar structures. Gummata in particular are not rare, ercles and gliomata ur. Above all it is nt to remember that ms of the basilar may play an impor- e, and give rise to ns like those of tu- one of the cranial But by far the most ng new growths in nity are those of the y body. Since the in close proximity optic chiasm, new involving this body e to a special set of ns. It is in these at we find the gen- aptoms of tumor as- with amaurosis and fore a distinct choked optic neuritis has leveloped. A pri- tic atrophy has also served in these cases. In consequence of pressure upon ic chiasm the amblyopia may be associated with temporal psia. The occurrence of amblyopia, temporal hemianop- h or without optic neuritis but associated with other symptoms pointing to intracranial tumor, may lead to picion of tumor of the pituitary body. This gland has years attained a considerable dignity in view of its supposed to acromegaly. That it bears an important relation to this can hardly be doubted; on the other hand it is but fair to at while simple hyperplasia, and occasionally a neoplasm ypophysis cerebri has been found in subjects of acromegaly, er of cases of tumor of the pituitary body have been recorded h there was not a single trace of any of the symptoms of lition known as acromegaly. Diabetes mellitus and polyuria

Fig. 35.—Round-celled Sarcoma of Hypophysis Cerebri. (After specimens and photographs prepared by Dr. Mandelbaum, pathologist to the Mt. Sinai Hospital.)

have also been observed as occasional symptoms of tumor in this region. The development of an enormous hydrocephalus, as determined by Roth and others, is not surprising in view of the peculiar situation of this gland (see Fig. 36).

Tumors in the vicinity of the sella turcica will cause very much the same symptoms as those which we have just attributed to the pituitary body, but the symptoms of acromegaly seem never to be

FIG. 36.—Same as Preceding in Longitudinal Section.

present unless the gland itself is involved in the new growth. If the growth in the anterior cranial fossa invades the orbital fissure we are very apt to have symptoms pointing to paralysis of the various ocular muscles as well as an exophthalmus which is generally unilateral. In some instances the tumor may invade the nose or the nasopharynx and actually give an opportunity for microscopical examination by removal of the new tissue which may be within reach. Unilateral anosmia, unilateral amblyopia with atrophy of one optic nerve, ocular palsies, paræsthesiæ in the division of the first trigeminal branch, and exophthalmus are the most reliable symptoms of tumor in the anterior fossa pressing upon the base of the frontal lobe. From what has been said regarding the frontal lobe we may also infer that in these cases mental changes, stupor, and even dementia are not infrequent. As the influence of the tumor is increased caudad, motor symptoms,

convulsions, hemiparesis, and even pain in the opposite half of the body may be added.

Tumors of the middle cranial fossa will be distinguished from the others by the involvement of the third, fourth, and fifth cranial nerves and particularly by its effect upon the Gasserian ganglion, in which case all the three branches of the trigeminal may be affected. There may also be involvement of the third and sixth nerves and the clinical picture may be complicated by involvement of the optic tract as well as of the chiasm.

If the tumor is situated in the posterior cranial fossa the symptoms will be like those due to disease of the pons. If there is any one symptom which will help to distinguish between these osseous growths and tumors of the pons itself, it is the exaggerated painfulness upon percussion of the skull. It has also been noted that in such cases pressure exerted upon the bony parts through the mouth and pharynx is extremely painful. According to the exact extent of the growth and the direction in which pressure is exerted, the symptoms may point to involvement of the pons, of the medulla oblongata, or of the cerebellum; but there is no need of repeating again the several sets of symptoms which will be produced by involvement of these various parts, nor is there any great practical importance to be attached to such minute differential diagnosis, for all of these tumors are unfortunately inaccessible to the surgeon.

Finally it is well to note that the attempt has been made to obtain other symptoms which shall point to the presence of intracranial tumor and possibly to its more precise location. The painfulness to percussion has been noted before, and in every case in which intracranial neoplasm is suspected such percussion should be carefully made. If there is a well-circumscribed area of pain, such evidence has at least the value of corroborating the local diagnosis obtained from other symptoms; but painfulness on percussion is a symptom by no means peculiar to tumors, and is found with much more frequency in cases of chronic meningitis such as is associated with syphilis. The temporal bone is peculiarly sensitive to percussion, and this fact should be borne in mind if mistakes are to be avoided.

The attempt to develop the art of percussing the skull has up to the present time yielded scanty results. Macewen's symptom has attained a certain fame within recent years. He describes it as the elicitation of a differential cranial percussion note as an aid to cerebral diagnosis. The percussion note "is obtained by the cranial walls vibrating when struck, the note being modified by the consistence and volume of the contents and their relative position to the bones. . . . The note is best elicited near the pterion, or a little posterior to that

point, but when distinct it may be produced all over the parietal and frontal bones." It is based upon the supposition that the ordinary percussion note becomes much more tympanitic if tumor is present; but this symptom, even if present, is to be used with the greatest possible caution because it can be obtained in other conditions in which there is a marked increase of intracranial fluid, or in which there has been a thinning out of the cranial bones. In making this percussion it is well to bear in mind that the thickness of bones in different skulls varies very much, and that little is to be gained by noting a difference in the percussion note until we have succeeded in comparing the percussion notes of a very large number of heads, or unless a distinct change in the percussion note can be elicited in one and the same patient at different times during the progress of the disease. The note can be elicited best by tapping the skull lightly with the finger or percussion hammer and receiving the note through a stethoscope placed upon the middle of the forehead or some point near the middle of the skull. Bruns seems to attach considerable importance to this examination, but up to the present time it has been of comparatively little service in my experience. The cracked-pot resonance has been noted by some, and Bruns states that he has been able to observe it in children under twelve years of age. This is probably due to remarkable thinness of the cranial lobes, and does not necessarily indicate the presence of any morbid process. Auscultation of the skull has been practised, and it is claimed that aneurysm of the basilar arteries can be detected in this way. Oppenheim states that blowing sounds which can be heard are not a safe sign of aneurysm, for they occur if vascular tumors are present and can be perceived in young children under normal conditions if the large fontanelle is not closed. He also ventures the statement that similar sounds can be preceived on auscultation of the skull in cases of extreme anæmia in the adult, but this observation seems to me to need further corroboration. In order to get the auscultation note the patient must stop breathing, and the examiner must pay the closest possible attention to the perception of such sounds. By pressing on the carotids Oppenheim claims that the sound varies and that, when the circulation is uninterrupted, it becomes more distinct.

Mills and Lloyd, and we believe Gray also, have attached some importance to variations in the temperature of the head in cases of tumor, and to the fact that such temperature was supposed to be greatest in a portion of the skull corresponding to the site of the tumor. This sign has never been of the slightest value to me and it is doubtful whether the observations are correct.

Differential Diagnosis.

In view of the great multiplicity of symptoms which may be produced by intracranial neoplasm, it is easy to see that if we wish to be explicit we might have to consider many of the diseases of the central nervous system, both organic and functional. Confusion has arisen not only with multiple sclerosis and with specific endarteritis, but also with conditions of neurasthenia and hysteria. But the latter need hardly be considered, though I may make reference to a single case, that of a man who was guilty of sexual excesses who complained of occipital pain radiating into the spine, of general lassitude, and who exhibited depression; the symptoms were at first thought to be due to a condition of mere neurasthenia, but the further progress of the disease proved conclusively that his symptoms were those of an intracranial neoplasm. We cannot take the space to allude to all such possibilities. The diseases with which tumor of the brain is most frequently confounded are abscess, meningitis, and hydrocephalus. The differential diagnosis between tumor of the brain and abscess will depend largely upon the presence of fever, upon the slow invasion and the slow development of all the symptoms in the latter condition, upon the occasional absence of optic neuritis in spite of the presence of symptoms pointing to increased intracranial pressure. But as all these symptoms are variable both in tumors and in abscesses, it is my own belief, based upon a fairly large experience, that the question will generally have to be decided upon the presence or absence of those conditions which predispose to the development of abscess. The difficulties of the differential diagnosis will be appreciated if we state that others as well as myself have observed cases of abscesses in which there has not been a particle of fever from beginning to end, and I can recall a patient who for months was lying in the hospital with multiple abscesses of the brain, some of them opening outwards, and yet fever and chills were only rarely present. The same variation in the occurrence of optic neuritis adds to the difficulties of the differential diagnosis. In a recent case I felt certain that the presence of slight fever, and the entire absence of optic neuritis argued in favor of abscess, but the post-mortem examination revealed a tumor in the temporal lobe. However much we attempt to differentiate between the two conditions, the difficulties are extremely great, and this need not be surprising if we remember that but for its infective character an abscess presents the mechanical conditions of a tumor. Oppenheim seems to me to underrate the difficulties of differential diagnosis; yet his statement regarding the ease of differ-

entiating between abscess and tumor seems to be based chiefly upon his dependence upon the preceding etiological conditions. If the symptoms come on after otitic disease or after traumatic injury, or if there is a febrile condition present, the diagnosis of abscess is, of course, easy enough; but it must be remembered that traumatism is a very frequent etiological factor in tumor, and that, on the other hand, abscesses commonly result from traumatic injuries which were so slight as to have escaped notice. The points which will help to differentiate between the two diseases are, at least in quickly developing abscesses, a rapid increase of the stupor leading to complete coma, and the much more rapid development of the focal symptoms. But chronic abscess is quite as frequent as acute abscesses are, and in the former all the symptoms may be developed with extreme slowness. If we are to distinguish abscess from tumor we are, therefore, compelled to depend chiefly upon a preceding otitis or some other lesion predisposing to the development of the abscess, upon changes in temperature, and upon an unusually rapid development of all the symptoms. But, as has been noted before, each one of these symptoms may be absent.

The ordinary forms of meningitis are so distinct that they cannot be readily confounded with tumor of the brain; but the differential diagnosis from tuberculous meningitis is made more difficult by the fact that some of these forms are developed very slowly and that the development of solitary tubercles may be associated with a meningitis. In meningitis, whether of tuberculous character or not, rapid involvement of a number of cranial nerves without an increase in all the other symptoms will argue in favor of this disease rather than of tumor. This is true both of the specific and of the tuberculous form of meningitis. If there is reason to suspect specific trouble it must be borne in mind that the formation of tumors, gummata, is quite common, together with the development of a specific meningitis. Apoplectiform seizures occur which are supposed to be due to the ordinary vascular accidents, but are in reality the expression of an invasion of a new growth into the motor area, or of a hemorrhage from a neoplasm. Such cases can generally be recognized by the fact that general symptoms, occasional vomiting, headache, etc., have preceded the onset of the apoplectic attack, and by the very different behavior of the paralysis if such paralysis is due directly or indirectly to the presence of tumor. A few months ago I had occasion to see a patient in a hospital, whose hemiplegia remained complete for a number of weeks without showing the slightest sign of improvement and whose general cachectic condition attracted my attention. On further inquiry I was told that she had been feel-

ing poorly for a number of months before the attack, suffering much from vomiting, dizziness, and intense headaches. An examination of the eyes revealed double optic neuritis, and in view of all the circumstances it was easy to make a diagnosis of intracranial neoplasm. This diagnosis was corroborated by a post-mortem examination which revealed a large sarcoma in the centrum semiovale.

Another condition easily confounded with tumor is that of hydrocephalus internus. The difficulties of differential diagnosis are increased by the fact that hydrocephalus internus is a frequent complicating condition of intracranial neoplasm and it is not easy to determine in advance whether tumor is or is not present. All the symptoms of intracranial pressure may be present in cases of hydrocephalus; yet some reliance may be placed upon the fact that hydrocephalus must attain an enormous development before it leads to optic atrophy and blindness. The general configuration of the head so characteristic of hydrocephalus is wanting in cases of tumor, and the development of all the symptoms is not nearly so rapid in hydrocephalus as it generally is in solid new growths.

We need not specially consider the differential diagnosis between tumor and dementia paralytica except to say that slight mental changes, apoplectiform and epileptiform seizures occur in tumors of the brain as well as in general paralysis, but the insidious onset of all the symptoms in general paralysis will help to distinguish between it and cerebral tumor; moreover, the ocular symptoms present in the earliest stages of general paresis are very different from those present in intracranial neoplasm, and this leads to a final remark regarding the value of optic neuritis to which so much importance is generally attached in the diagnosis of tumor. I have known the diagnosis of tumor to be made simply upon the presence of this one symptom, for the fact is not generally appreciated that optic neuritis is present in a number of different diseases. First of all, it seems to me to be more frequent than it is generally supposed to be in syphilis of the brain. As these cases are often associated with headache, with epileptiform seizures, sometimes with slight mental changes, it is easy to see that a confusion may arise. Optic neuritis is undoubtedly present in some forms of multiple neuritis and it is also produced by a number of toxic substances. If we finally concede its frequency in different forms of meningitis and in encephalitis it will be clear that the diagnosis of tumor should not be made unless this one symptom is associated with other characteristic signs of intracranial neoplasm.

If we have succeeded in establishing beyond reasonable doubt the presence of tumor, the greatest care must be exercised in considering the focal signs, so that if an operation is undertaken serious error

ment of tumor and occasional apoplectiform seizures argue in favor of gliomata. These are very frequent, too, in children, and since they occur most commonly in the cortex and in the cerebellum, all the attending circumstances of the case may assist in making a correct diagnosis. A marked remission in all the symptoms is supposed to argue chiefly in favor of gummata or possibly of an aneurysm; but such a cessation in the symptoms, as we have seen before, may occur with glioma and has also been reported in cases of solitary tubercle. While a fair inference may be allowed from the results of antisyphilitic treatment, too much dependence cannot be placed upon this one fact. The remission in the symptoms may be due very largely to absorption of part of the hydrocephalic fluid which is present in so many cases of intracranial neoplasm.

Course and Prognosis.

The majority of tumors of the brain are developed in a very insidious fashion. As a rule slight headaches and occasional vomiting, or twitching movements so slight that they are not suspected to be of any serious moment, precede the onset of other symptoms for a period of several weeks or months before the true character of the morbid process is suspected. There are exceptions, however, to this rule, and in some instances the symptoms are developed so rapidly that an acute inflammatory condition seems more likely than a solid neoplasm. The suddenness in the development of the symptoms must be attributed either to a rapid increase in the growth of the tumor or to the occurrence of a hemorrhage into the tumor. Such hemorrhages are not infrequent in the course of development of a glioma. If the tumor is of a cystic character an increase in the fluid contents may give rise to an acute set of symptoms. It is also to be noted that tumors occurring in the silent areas of the brain may give rise to few or no symptoms, but that as soon as they reach the motor area or some other area with distinct functions the symptoms pertaining to disease of these areas are developed more or less acutely.

Intermissions and complete remissions occur with tumors of all sorts. In the case of syphilitic neoplasms such remission which may possibly lead to a recovery can be readily understood, but the fact that similar remissions may occur with gliomata and even with solitary tubercles, is not so easy to explain except on the supposition that for some reason or other the growth of the tumor is inhibited, and as in the case of solitary tubercles the tumor may become encapsulated and thus act as a non-irritating foreign body, causing no or at least very few symptoms for a prolonged period of time. I have several

times referred to a very marked instance of a prolonged remission in a man of forty odd years who had had occasional epileptic seizures, had suffered an attack of hemiplegia in earlier years, and had been troubled greatly with headaches. A specific infection had been conceded by the patient, and the diagnosis of a specific neoplasm was by other physicians as well as myself thought to be probable. The patient had been placed on the inunction and iodide treatment and had improved to a very remarkable extent so that he appeared to be entirely well for several months. Suddenly an apoplectic seizure occurred and death ensued twenty-four hours afterwards. The post-mortem examination revealed a glioma of very considerable size involving the arm and face centres of the right side, and it was of a size which made it probable that the tumor had been in existence for several years. Whether the remission was due to an arrest in the growth of the tumor or to a change in the amount of cerebrospinal fluid it is difficult to determine; and this variation in cerebrospinal fluid and the change in the symptoms depending upon such variation have to my mind not received the attention which they deserve.

Tumors of the brain are almost invariably fatal. The cases which I have seen have run their full course within a period of two years. Others have reported cases which have continued to live five and even ten years after the initial symptoms have been observed, but unless such observation is verified by a post-mortem diagnosis I should be inclined to suspect a mistake in diagnosis rather than that the tumor had actually existed for such a period of time. I myself made the diagnosis of tumor of the brain in a man of thirty more than three years ago. The headaches were extremely characteristic; there was and is double optic neuritis, and while all these symptoms have persisted since the beginning, I feel more inclined to suppose that there is some other morbid process present than to insist that the case is undoubtedly one of solid tumor. After the affection has run a course of one or two years the patients are apt to die of some intercurrent disease or of some one of the many complicating conditions. Sudden death occurs as a direct cause of the tumor with greater frequency, at least in my experience, in the case of cerebellar tumors and new growths in the posterior cranial fossa than in any others. That tumors may undergo spontaneous cure can hardly be doubted, although the occurrence is rare enough to make it of little practical value. Such cases have been observed by Bruns, Gowers, Oppenheim, and others. Aneurysms and ecchinococcus cysts are among those which undergo spontaneous cure or in which there may at least be a complete arrest of growth. Solitary tubercles may share the same fate, but it is the rarest of all occurrences. Finally it is im-

portant to note that the double optic neuritis to which so much importance is attached may persist for a long period of time after every other symptom of neoplasm has subsided. I have observed for many years a lady whose health has been exceptionally good, whose past history is perfect, who has never had any miscarriages, but who developed at least four years ago a double optic neuritis of mild degree which has neither progressed nor receded. The optic neuritis was corroborated by one of our best oculists. While both he and I suspected tumor many years ago, we are now certain that the case must have been one of gumma, and that we must regard the optic neuritis as the sole residue of a former specific cerebral trouble. After all the data have been reviewed we stand face to face with the fact that surely ninety-five per cent., if not ninety-nine per cent., of all cerebral tumors end within a few years in death.

Treatment.

Considering the unfavorable prognosis in cases of cerebral neoplasms, it would be fortunate indeed if we could hope for marked success from the treatment of these conditions. Long before the beginning of the era of brain surgery various attempts had been made to inhibit or retard the growth of brain tumors. The success of such efforts has been slight indeed, and yet even at the present day physicians feel compelled in every instance to attempt medicinal treatment before proposing or resorting to surgical measures. This is done in obedience to medical prejudices and not from the conviction that good results are likely to follow upon the use of purely medicinal measures. The only tangible result is that the physician's conscience is satisfied that he has not exposed the patient to a dangerous operation without having given him the chance of relief by other measures. But why should we hesitate to suggest surgical remedies if we know by experience that all other measures of relief have proved entirely unsatisfactory? It is more than probable that valuable time has often been lost by this procedure and yet few physicians will be willing to adopt any other course. My own experience has taught me that even at the risk of a possible mistake in diagnosis a trephining operation in the earliest period of the disease may be entirely justifiable. Thus in one case in which a tumor was suspected, because, in addition to other symptoms, a young man of about twenty years had presented sudden twitching movements in one upper extremity, I agreed to the wisdom of an immediate operation with the view of determining whether neoplasm was present or not. In that case the operation showed that there was an area of traumatic encephalitis, and the ex-

cision of that area was sufficient to cure the focal epilepsy from which the boy was suffering. Had there been a small tumor in the same area the operation would have been equally justifiable and of even greater benefit. I am not pleading for indiscriminate surgical practice, but I do wish to insist that if the symptoms are of such a character as to render the diagnosis of tumor probable, and if they point to a well-defined area of the brain, an exploratory operation should be attempted, for there is little danger in such operations. Much good may come from such surgical practice and very little harm, while the practice of deferring surgical interference until other measures have proved fruitless implies or means the loss of valuable time—the time during which the tumor may have grown to such an extent as not to be capable of removal. Bruns, who takes very much the same view of the situation and whose experience has perhaps been the largest among German neurologists, is willing to agree to a period of six weeks during which medicinal treatment shall be given. I am willing to agree with this author that six weeks should constitute the extreme limit of time; but knowing how little can be accomplished by medicinal treatment, I should be in favor of operating even earlier and of operating as soon as the diagnosis of an operable tumor is made. The only medicinal treatment that has proved to be of any account is the thorough course of iodides or of inunctions of mercury, and the one reason why we still cling to this method of treatment is that there is always a little doubt as to whether or not the tumor in a given case may be syphilitic and therefore amenable to treatment. The temporary success following upon such a course of treatment has been the source of errors in diagnosis, for, as we have stated before, an improvement in the symptoms may ensue even though the tumor continues to grow; and in distinctly specific growths the iodides and mercury are often of little use.

I have thought it best to weigh the advantages of medicinal and surgical treatment at the outset; but the question now arises whether the results of surgical treatment have been such as to encourage us to recommend surgical interference in many cases. In attempting to decide this question let us note first of all that only a relatively small percentage of all the cases of brain tumor are proper subjects for operation. Ever since the successful work of Horsley had been made known, cases of brain tumor that seemed to be within reach of the surgeon's knife have been operated upon, and yet the actual percentage of tumors that have been or could have been removed remains extremely small. In a series of one hundred tumors in the museum of Guy's Hospital, Hale White found ten which might have been removed safely. Mills and Lloyd also state that in their collation of one

hundred cases there were ten which could have been operated upon. Knapp found but two in forty cases, and Dana states that in five of twenty-nine of his cases the tumor could have been removed. Starr, in analyzing six hundred tumors, states that thirty-seven, that is about six per cent., could have been removed, but these figures do not give a very accurate idea of the surgical possibilities of the future, for many of the cases were observed at a very late period when the tumors had attained enormous size and could not have been removed, whereas if the diagnosis had been made earlier the attempt at removal might have been successful. In spite of the unfavorable percentage of cases, it may be stated that every tumor which can be reached is capable of removal, and that tumors near the outer surface of the brain and not in silent areas are those which would properly come under the surgeon's care. For the present, tumors at the base of the brain and in the interior cannot be reached by the surgeon. The attempt has been made to remove cerebellar tumors, but so little success has been obtained that unless better methods are discovered we fear that they too must be included in the category of inoperable tumors.

In a total number of ninety-seven cases analyzed by Starr in 1893, eighty-one were tumors of the cerebral hemispheres, sixteen tumors of the cerebellum; the tumor was not found in thirty-five cases; in one case the cerebral tumor was found present, but was not removed, and the same was true of two cerebellar tumors. In thirty-nine cases of cerebral tumor the growth was removed and the patient recovered, that is, survived the operation; the same was said of three cerebellar tumors. The tumor was removed and the patient died in fifteen cases in which the cerebrum was involved, and in two in which the cerebellum was involved. A temporarily satisfactory result was therefore gained in only one-half of the ninety-seven cases, and if we represent to ourselves the fact that these ninety-seven cases would represent only about seven per cent. of all cases of cerebral tumors we can see that the outlook is not a very brilliant one. But within the past two years several notable instances of the successful removal of large tumors by Thomas and Keen and others have been reported upon, and however little promise statistics may hold out, it is clearly our duty to endeavor to attain further success in this field by greater care in the early recognition of the tumors and by greater skill on the part of the surgeon. The number of cases in which the tumor is found and removed but the patient dies soon after removal can probably be lessened very much. From my own experience I am convinced that cranial surgery must be practised with even greater care than has been the case hitherto and that above all the effect of shock from the operation must be

avoided. One element of danger is the excessive loss of blood and the large amount of time that is consumed in controlling hemorrhages. Unfortunately all surgeons have not adopted the practice of some, of encircling the head with some constricting ligature before the scalp is incised and thus avoiding the loss of a large amount of blood. In children in particular the shock of cranial operations is to my mind more largely due to the hemorrhage than to any other one factor, and this applies not only to operations for the removal of tumors, but also to those that are carried out for the cure of epilepsy.

If the removal of a large neoplasm seems impracticable at one sitting, the division of the operation into two or more stages might be resorted to. It is my firm belief, however, that if the surgeon will endeavor to cut off the blood supply of a certain area of the cortex before excising it even large tumors can be removed at a single sitting. Several operations may be necessary and justifiable as in a case of recurring gliosarcoma reported by Erb.

It is a fortunate matter that the tumors occur with relative frequency in the motor area, that these very tumors give rise to the most distinct set of symptoms, and that they are in that portion of the brain upon which surgical operations can be attempted with the greatest impunity.

Tumors in the occipital and in the frontal lobes could be reached easily enough, but the localizing symptoms are not always so clear as to justify surgical interference. In the case of tumors of the occipital lobes the matter is rendered a little more difficult by the fact that the tumor may be situated on the median surface and thus be inaccessible. It would be particularly desirable to be able to get at tumors of the cerebellum, but here the surgical difficulties are very great. The area in which the surgeon is bound to operate is so circumscribed, there is such great danger of profuse hemorrhage from the various sinuses, that the practical difficulties far outweigh the possibilities of success. In one case in which I agreed to the removal of a cerebellar growth, and in which the diagnosis could be made with the greatest ease on account of the presence of facial and auditory symptoms, Gerster made the attempt to reach the tumor, but it was found to be practically impossible to remove it. And it is in this special field of cranial surgery, that is, the removal of cerebellar tumors, that further studies are necessary. The point of entrance into the cranial cavity has not yet been satisfactorily determined. But even if the focal symptoms of a tumor are such as to point clearly to its seat, and if all other conditions would appear to be favorable for successful operation, there is one circumstance over which we have no control, which has been the cause of great disap-

pointment; this one circumstance is our imperfect knowledge regarding the exact size of a tumor. The focal symptoms are caused by the invasion of definite areas, say the motor or speech areas of the brain, but the tumor causing such symptoms may have started from a distance and if it has been of relatively rapid growth the neurologist or surgeon may not have suspected its origin, in the frontal lobe for instance, and may not be capable of forming a judgment as to the exact size of the tumor. The successes of Keen, Kocher, and others in removing large tumors lead to the hope that within a reasonable period of time the mere size of a tumor will not count so heavily as it has in the past against recovery after operation. But even the partial removal of tumors may be attended by temporary success. Dana exhibited a young patient before the New York Neurological Society who had done well for more than a year after the partial removal of a sarcoma. The character of the tumor also has a bearing upon the advisability of operation. It is an open question whether tubercles should ever be operated upon, but in several instances such removal has been attempted without the true character of the tumor having been determined in advance. Czerny, as reported by v. Beck, removed a tubercle and the patient was said to have done well after the operation. The multiplicity of tubercles and other malignant growths militates against the advisability of such operations; but if the patient has survived the operation he has surely been none the worse for it, although he may succumb to the malignant character of the disease later on.

It is also a question whether metastatic tumors should be included in the list of operable cases, but inasmuch as we do not hesitate to operate upon metastatic tumors in other organs, there is no reason why we should hesitate in the case of cerebral neoplasms. There is still another consideration which would appear to make these operations wholly justifiable. The patients endure great torture from excessive headaches, sometimes from persistent vomiting; they are suffering from a disease that is incurable in fully ninety-five per cent. of the cases, and their only hope is in operation. If they cannot be cured by the operation their symptoms can at least be relieved. Most of us have therefore been willing to adopt Horsley's suggestion that the operation is justified for the relief of headache, even though nothing else be gained by it. The creation of a large trephine opening lessens intracranial pressure, and if such a trephine opening be made in a part of the skull from which a hernia cerebri is not apt to protrude the condition of the patient will be much more bearable after the operation than it was before.

The evacuation of a cyst or the excision of the entire cyst has

been followed by good results. Such cases have lately been reported by Stieglitz and Gerster and by Eskridge and Parkhill. The introduction of a gold plate between the dura and scalp to prevent adhesions has been practised by Gerster and others, and is specially serviceable in those cases in which a second operation (refilling of a cyst) is to be taken into account.

Lumbar puncture as introduced by Quincke has been suggested as a palliative measure in the treatment of brain tumors. The withdrawal of a considerable amount of cerebrospinal fluid was supposed to lessen the headaches and other symptoms due to increased intracranial pressure. While I would favor cranial trephining in all cases in which it can be practised, Quincke's method may be adopted in such cases in which a cranial operation would seem unjustifiable or in which it is not permitted. The only class of cases in which I would distinctly caution against this procedure is in cerebellar tumor. I have had an experience corroborating that of Fürbringer, who found that sudden death occurred in several cases of cerebellar tumor after the removal of a considerable amount of cerebrospinal fluid. In a case which I saw in consultation about six months ago, in which the diagnosis of an inoperable cerebellar tumor was made and was corroborated by a post-mortem examination, death ensued twenty-four hours after the withdrawal of a few drachms of cerebrospinal fluid. Death was the result of interference with the cardiac and respiratory centres, and there is very little doubt in my own mind that in consequence of the withdrawal of fluid and the change of pressure the tumor had fallen down upon the fourth ventricle, and had acted as a ball valve. This danger has not been sufficiently realized and it is for this reason that a word of caution is urgently needed.

In concluding this discussion of the surgical methods for the removal of intracranial neoplasms we may say that in spite of the few successes that have thus far been obtained, the only hope for the cure of these neoplasms lies in surgical means. The failures of the past need not discourage us, but the neurologist and the surgeon must unite their efforts, the one in making an early recognition of tumor possible, the other in perfecting the methods by which such tumors are to be removed. There is very little doubt that if the matter is given careful attention, far greater success is to be expected within the coming decade.

NOTE.—Cranio-cerebral topography does not come within the scope of this article. The facts and methods which it is necessary to know may be gathered from the works of Chipault, of Broca and Maubrac, from Starr's "Brain Surgery," p. 15, or from the present writer's book on "The Nervous Diseases of Children," p. 448. I wish to call attention once more to the great practical value of making the elec-

trical tests upon the exposed dura or cortex, if the motor area be the site of the tumor.

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DISEASES OF THE MENINGES.

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DISEASES OF THE MENINGES.

MUCH confusion has arisen concerning meningitis because of the improper understanding of the anatomy and relationship of the coverings of the brain, the so-called meninges. Many modern textbooks of anatomy teach the existence of three membranes, a dura mater, a pia mater, and an arachnoid. A good many ingenious diagrams and illustrations are produced to demonstrate these, but they do not fulfil their purpose. As a matter of fact the membranes of the brain are two in number: the dura and the pia mater. The dura, a thick, resistant, fibrous membrane, consists of two layers. The outer is in intimate apposition with the inner surface of the skull, and serves as periosteum. The inner layer of the dura is closely united with the outer layer, but separated from it in many places to form channels for the passage of blood, the sinuses of the dura mater.

In addition to the sinuses the dura contains arteries, lymphatics, and nerves. It serves a double purpose, protective to the brain, protective and nutrient to the bones of the skull. At each of the foramina at the base of the skull it blends intimately with the margins, while sheaths of the pia enclosing the outgoing nerves pass through. A knowledge of this latter fact is of clinical importance, because not infrequently these vaginal prolongations of the pia are the pathways through which infection by contiguity arises.

The dura mater, a very fibrous and not very vascular membrane, is not prone to inflammatory diseases. In fact, diseases of the dura proper, aside from those caused by traumatism, are the rarest of intracranial conditions.

The pia mater is a highly vascular double membrane, the inner layer of which, properly called the visceral pia, is in intimate contact with every part of the cortex, between the fissures, at the bottom of the fissures and on the superficies as well. This vascular layer of the pia sends prolongations bearing blood-vessels into the substance of the brain, which communicate with the interventricular vessels and which are known as the tela choroidea. The external layer of the pia, which should be called the parietal layer, is laid loosely on this visceral pia and bridges the convolutions, but does not dip between them. Between the visceral pia and the parietal pia is the pial

space, the intermeningeal, the so-called subarachnoid space, constituted by lymph spaces and channels, in which the blood-vessels ramify and in which pathological products accumulate in disease states. It is thus seen that the pial covering of the brain is somewhat analogous to the pleural and peritoneal covering of the viscera. It fulfils a strictly analogous purpose, and its construction is on practically the same lines. The layer of the pia called the parietal is the one usually described as the arachnoid, and the space existing beneath the two layers is frequently known as the subarachnoid space; but, as we have intimated before, this is a needless and confusing use of terms which we shall endeavor to avoid as much as possible. The pial space, which, like the pleural space, exists under normal conditions largely in possibility, may in diseased conditions become enormous. The roof of this space is formed by the bridging of the sulci and the great fissures and of the spaces existing between the component of the parts of the brain by the parietal or arachnopia, while the floor is formed by the visceral pia. This space is normally bathed in lymph, which is in communication with all of the lymph channels and spaces of the other parts of the nervous system. The communication is effected by the Pacchionian bodies, extending from the parietal pia through the subdural space, and by the external sheaths of the pial blood-vessels, which project down through the substance of the brain and in through the ventricles. The pial spaces of the brain and cord—what are called the subarachnoid spaces of the brain and of the spinal cord—are contiguous; that is to say, they form a continuous space separated by constrictions, and may be emptied or filled one from the other. It is important for the proper understanding of pathological conditions in inflammation of the pia, that it be kept in mind that there is nothing between the parietal pia, the so-called arachno-pia, and the dura, but that there exists a subdural space which is the seat of disease products in inflammation of the dura mater.

MENINGITIS.

Inflammation of the pia mater, leptomeningitis, is by far the most frequent and most important inflammatory condition that occurs within the cranial cavity. It is the process that is meant when the term meningitis alone is used. In the present stage of the rehabilitation of the pathology of inflammation it is not easy to give the best classification of leptomeningitis. It is needless to say that the trend of pathology to-day is to predicate for all inflammatory conditions the existence of vegetable organisms, although it still seems necessary

to admit that the products of such organisms may cause inflammation without the organisms themselves being present.

Leptomeningitis might well be classified as of endogenous and exogenous origin; grouping under the first heading all those conditions, capable of giving rise to the disease, which develop within the body; and under the latter, all the morbific conditions that have their origin without the body. I prefer, however, to classify meningitis according to its causation and pathological products, and shall speak of it as—

I. True, or genuine meningitis.

II. False, or pseudomeningitis

True meningitis, or leptomeningitis, is subdivided into:

1. Leptomeningitis, whose pathological product is of a serofibrinous, seropurulent, or saniopurulent nature, depending upon the kind and intensity of the bacterial infection, but which always contains pus in greater or lesser quantity. Topographically the exudation is predominantly over the convexities of the hemispheres. Bacteriological examination reveals the presence of some pyogenous organism, the pneumococcus, the streptococcus pyogenes, the diplococcus pyogenes, the bacillus of typhoid fever, the specific organism of ulcerative endocarditis, of gonorrhœa, or of erysipelas, or some pyogenic organism not yet satisfactorily classified, such as the bacillus of Neumann and Schaeffer.

2. Cerebrospinal meningitis, a specific infectious form of meningitis, which prevails epidemically and sporadically, whose pathological product is not materially different from the ordinary form of meningitis. Topographically the lesion is distributed over the meninges of the entire central nervous system, sometimes predominating in the meninges of the cord, while at other times the pial covering of the brain bears the brunt of the disease. Bacteriological examination reveals the presence of two different organisms, which are considered to stand in causal relationship: the diplococcus intracellularis, so called because it is found within the body of the cells, which is believed to be the real bacterial cause of the disease, and the diplococcus of pneumonia.

3. Tuberculous meningitis, characterized particularly by the eruption of tubercles and of a serous and serofibrinous exudation, but which is often accompanied with a purulent exudate, the result of the pyogenous properties of the tubercle bacillus in some cases, but more often the expression of a mixed infection. The eruption of tubercles occurs so frequently in the meninges of the base of the brain that this location has come to be looked upon as characteristic.

False or pseudomeningitis, a condition which is designated mé-

ningisme by the French, a name which I trust may never be added to English nomenclature, really needs no subdivision. In order, however, to facilitate the recognition of clinical types I shall refer to it as—(1) alcoholic meningitis, a dropsical condition of the meninges and brain occurring in chronic drunkards and commonly known as wet brain; and (2), serous meningitis of infants and also of adults, an expression of a diathetic and depraved state which sometimes follows exhausting diseases and rheumatism.

Personally I share the opinion of those who believe that the time has come to classify meningitis strictly according to its bacterial cause. Clinically, however, there is not yet sufficient justification, and I shall content myself with the mention that it may be classified according to its causation by—(1) cocci, (2) bacilli, (3) polymorphic bacteria, or (4) a mixed infection. These various bacterial causes will be considered in some detail in the section on bacteriology and pathology.

Syphilitic leptomeningitis in the strict interpretation of the term—*i.e.*, true inflammation of the meninges—does not exist unless one is willing to admit, as some are, that occurrence of exudation is synonymous with inflammation. Syphilitic infection may produce disease of the meninges in two ways: First, by causing a degeneration of the pia, a condition mediated through its vascular supply, and second, by influencing the pia so that it becomes prone to the action of those factors which produce inflammation.

Like every other disease of serous membranes, leptomeningitis may pass into a more or less chronic condition, which is always, however, secondary to an acute process, unless the change in the meninges be a degenerative one. Chronic meningitis, with the exception of the syphilitic variety, occupies a very unimportant place among the intracranial diseases; it will be referred to in the section on the syphilitic variety.

I propose to consider meningitis clinically from a somewhat different standpoint than that ordinarily taken in the books. In the latter we hear very much of refinements in differentiating clinically the various forms of meningitis. At the bedside, however, we know from experience that it is next to impossible to make a distinction between any of the forms except to differentiate the tuberculous. I intend, therefore, to give a general description of a case of leptomeningitis of ordinary severity, in which pus is one of the pathological products, and then to consider the factors that enable us to make a more specialized diagnosis, and especially to point out how the epidemic form, although caused by and associated with specific organisms very similar to the ordinary form of purulent meningitis,

is often attended by such striking symptoms as to constitute a definite clinical entity.

The clinical forms of pseudomeningitis will then be considered particularly in reference to differential diagnosis.

Leptomeningitis.

History.

The history of leptomeningitis is veritably an interesting one, and a simple retrospect of it will show the great advance made during the last few years concerning the nature and causation of this affection. It is probable that many of the cases described under the name of frenzy by the ancient writers were examples of some form of meningitis, but the first recognition of meningeal disease as an entity was made by Robert Whytt, of Edinburgh, whose observations on dropsy of the brain were published posthumously, in 1768. Up to that time the term hydrocephalus was applied to almost every form of intracranial disease except hemorrhage. Previous to the time that hydrocephalus was recognized as a pathological condition, many of these cases were referred to as hypertrophy of the brain, and Morgagni gives a very detailed description of several such cases. Inflammation of the brain substance was not considered apart from inflammation of its coverings, until such a differentiation was suggested by Herpin, who first made use of the term meningitis. After the paper of Whytt, which was based on a study of twenty cases, in ten of which an autopsy had been made, a number of observations were reported by Fothergill, Ludwig, and others, but these authors made no endeavors to determine the pathogeny of the disease and looked upon their cases as examples of dropsy. Thus it was not until 1815, when Golis showed that ventricular dropsy was a secondary condition depending upon previous inflammation of the membranes or vessels of the brain, that attention was first called to a differentiation of intracranial inflammations, although Quin, and Ford in 1780, had expressed an opinion that the intracranial accumulation in hydrocephalus may have resulted from congestion. After the article of Golis, observations were published by Coindet in France and Abercrombie in England (1817), but it was not until 1825 that the term meningitis was employed by Senn to designate the condition which preceded the dropsy in such cases. In 1827 Guersant expressed the belief that the lesion of these cases, and which had been termed meningitis by Senn, was a form consisting of little granulations, and he therefore termed it granular meningitis, a designation which is still sometimes used.

A year or two later, two of his pupils, Demongeot and Confebron, went a step farther and associated the granular deposition with tubercle; although attention had been more widely called to this view by another pupil, Papavoine, who described two cases of arachnitis of tuberculous origin in 1830. Three years later Gerhard, of Philadelphia, published a remarkable paper, in which he detailed the result of thirty-two autopsies. It is because of the articles of Papavoine in France, and Gerhard in this country, that the profession soon came to look on inflammation of the meninges as examples of meningeal tuberculosis. Even after the large number of observations which were soon published on this disease, discrimination was not made between tuberculous and other forms of leptomeningitis until it was pointed out by Guersant, in 1839, and more vigorously insisted on by Barthez and Rilliet, in 1843.

The views expressed by the last two writers were not quickly accepted, and Watson in his lectures on the Practice of Physic, which were revised in the year 1843, speaks of the disease in the most obscure way. Since that time, however, until the epoch of the present generation the progress of differentiating the different forms of meningitis went on, until finally bacteriology revealed, through the instrumentation of Leyden (1883), of Saenger, of Weichselbaum, of Netter, of Flexner and Barker, of Foà and Uffreduzzi, and of innumerable other workers, that different forms of meningitis were different kinds of reaction to specific organisms.

Formerly the term tubercle was applied to all forms of nodules whether large or small, and the designation of the granulations in meningitis as tubercles carried with it its present significance only after Koch, in 1882, demonstrated the presence of the tubercle bacillus in all forms of exudative tuberculosis, and the dependence of them thereon.

The bacterial origin of epidemic leptomeningitis was shown first by Leyden, in 1883, who demonstrated a diplococcus in the cerebrospinal fluid and the tissue of the pia, which Fraenkel, and later Hauser, showed to be identical with the pneumococcus.

In 1887 Weichselbaum showed the presence in the middle of pus corpuscles from epidemic cerebrospinal meningitis exudate a diplococcus, which on account of its nidus is called the diplococcus intracellularis. Pio Foà and Guido Bordoni-Uffreduzzi later described a third and a fourth form of meningococcus.

In 1884 Adenot in France, and Weichselbaum in Germany, showed that meningitis which occurred during or after an attack of croupous pneumonia was dependent upon the same microbe as the pneumonia, a contention which is now universally accepted. Since

that time a great number of writers, among whom may be mentioned Grasset, Ortmann and Samter, and particularly Netter, have furthered our knowledge of the bacteriology of the forms of meningitis due to the action of cocci, and have particularly pointed out the occurrence of pneumococcal meningitis without antecedent or coexistent pneumonia. Grasset has shown, for instance, that meningitis occurring with inflammatory rheumatism may be dependent upon the pneumococcus; Gabbi and Puritz have found the same cocci when the meningitis was associated with periarthritis and endocarditis, and Ellehorst found them in a case of meningitis which was apparently due to fracture of the base of the skull.

Further details of the bacterial origin of meningitis will be given in the section on bacteriology and pathology.

Symptoms.

The symptoms of all forms of acute meningitis are somewhat similar. They vary greatly in individual cases, probably according to the different bacteria that produce them. In the following description a fairly typical case of acute coccal meningitis is kept in mind.

The symptoms of acute meningitis are usually divided, according to their predominant characters and the time of their occurrence, into three groups or stages: the first that of headache, in which the inflammatory condition is at its height, the second the period of delirium, and the third the stage of coma—the latter corresponding to the time in which the exudation and increase in interpial and interventricular fluid make serious pressure on the brain. This subdivision is very artificial, and we shall consider the symptoms under two headings, those occurring during the period of excitation and those present during the period of depression.

The onset of an attack of meningitis varies somewhat according to the exciting factors. Meningitis due to a not very virulent infection will be of very much less sudden and severe onset than one due to a pneumococcus or streptococcus infection. After the disease really begins there develop three symptoms at about the same time and with about the same degree of severity which at once suggest the disease. These are headache, vomiting, and constipation, and their onset indicates the beginning of the first period, the period of excitation. The pain in the head may at first be localized to the forehead, to the vertex, or to the occiput, but generally it is diffused and extends down the back of the neck and between the shoulders. It is intense, lancinating, and tearing in character. It increases in severity from hour to hour, until finally it becomes so severe that it

frenzies the patient and later submerges all the intellectua .aculties. It is aggravated by light, by sounds, by agitation, by anything that requires mental or physical action. The simple acts of coughing and sneezing, for instance, will so aggravate the pain even in the beginning of the disease that the patient avoids them at any hazard. The exacerbation of the headache by light gives rise to the symptom known as photophobia, a condition which is prominent from the beginning. In a similar way can be explained the increased sensitiveness of the hearing apparatus. In the very beginning there is a fixity about the head and the neck, which is probably partly voluntary on the part of the patient in order to avoid exacerbation of pain through agitation of the head, but this fixation soon becomes an involuntary symptom and a very prominent one. The increasing severity of the headache prevents rest and sleep, and provokes the most agonizing cries, which the patient may attempt to smother, but which in tuberculous forms of meningitis, occurring as the latter does with preponderating frequency in young children who make no attempt at repression, has assumed considerable symptomatic importance and is known as the hydrocephalic cry.

Vomiting, which occurs almost from the very onset of the disease, has the characteristics which have caused it to be known as cerebral; that is, it is not preceded by nausea; diaphragmatic action is extremely forcible, and expulsion of the contents of the stomach correspondingly projectile. Complete emptying of the stomach in no way relieves the desire. Efforts of vomiting are continuous, and, unlike gastric vomiting, this is entirely unamenable to any form of local treatment. The vomited matter itself consists naturally of any contents which the stomach may have; if it has none, of fluids stained with bile. Very rarely, almost never, is there anything like stercoraceous vomit. With the vomiting there develops the most obstinate constipation, for which no amount of therapy directed towards its relief is of the slightest service. This is accompanied, frequently from the beginning, with retraction of the abdomen, which has in some cases a very characteristic boat-shaped appearance. This is more frequent in the tuberculous than in the simple purulent form, and more frequent in the text-book than at the bedside. The retraction of the abdomen may last for several days, during the entire period of excitation in fact, and is followed oftentimes in the period of depression by extreme distention, tympanites.

It depends very largely upon the causation of meningitis whether or not there will be a high degree of febrile manifestation. If the cause be a profoundly septic one, the elevation of temperature following a more or less prolonged chill may amount to as much as

105° or 106°. As a rule, however, except in the beginning and in young children, the temperature very rarely will exceed 103°. The temperature range is not characterized by any regular exacerbations or remissions; there is generally during the period of excitation or increasing headache a rather constant and continuous fever. Sometimes during one of the violent exacerbations of pain and agonizing cries the temperature becomes suddenly increased.

Associated with the febrile manifestations, and progressing with them hand in hand, are certain vascular phenomena, the more prominent of which are paleness and mottling of the skin, contraction of all the peripheral blood-vessels, and increased tension of the large blood-vessels, which gives a hard, resistant, tense feeling to the pulse, which at the same time is increased in frequency. A sphymographic tracing made of the pulse at this time shows a very abrupt upstroke, a sharp apical marking, and an acutely beginning downstroke, which, after it passes the dicrotic notch, spreads itself out before it reaches the base level.

Symptoms of motorial irritation are prominent from the very beginning. It has been said above that even from the first pang of headache there is a certain immobility of the cephalic extremity which is more or less voluntary, but the disease has not become thoroughly established before there can be noticed a beginning involuntary fixation of the head associated with retraction of the neck. This fixation may go on to such a degree that the patient can be lifted by the head with the heels as a point of support, while the retraction may increase even to severe opisthotonos. Concomitantly with these developments there may be such universal symptoms of motorial irritation as twitchings or spasms of muscles or groups of muscles, fibrillary twitchings, and in children tonicoclonic convulsions of great severity. These phenomena of irritation may manifest themselves in any motor part of the body, but it is particularly when they show themselves in the distribution of some one or more of the cranial nerves that they assume a deeply significant import. If the meningitis be of the convexities it is natural that symptoms of motorial irritation will be earliest and most pronounced in the extremities, but if the inflammatory exudate be at the base and the basal convexities, which it is in the tuberculous form, symptoms of cranial nerve involvement will be early and pronounced; and of these latter none is of greater significance than strabismus, a deviation of the ocular axes due to spasm or contracture in one or more of the eye muscles. Although spasm and contracture occur much less frequently in the muscle supply of other cranial nerves, there sometimes occurs twitching of the muscles of the face, showing involvement of the seventh nerve,

which gives rise to grimaces and the so-called sardonic smile, and in the musculature of the ninth, tenth, and eleventh nerves which causes difficulty in swallowing, in phonation, and in articulation. Later on in the disease all these nerves may be partially paralyzed.

If now a general survey is taken of the patient, it is found that the face is suffused with colors of a primary lividity; the eyes are scintillatingly bright with injected conjunctivæ; the pupils are small, regularly contracted, at least in the beginning and respond very slightly, if at all, to light or in accommodation. The color of the skin and the entire body is red or alternatingly pale and red, due at first to a severe vasomotor contraction and later to corresponding paresis. When the finger or nail is drawn over the skin, a bluish-white line with purplish-red margins remains, which is known as the tache cérébrale, originally described by Trousseau, who, as well as many followers, attached an undue diagnostic importance to it. The respirations are accelerated in frequency and the rhythm of the inspiratory and expiratory wave is disturbed. They partake of the same motorial irritation that is manifest in every part of the body. The tongue is not infrequently foul and coated, although it may remain perfectly clean during this entire stage. The vesical sphincter is almost always derelict in function. Oftentimes from the beginning, and particularly if the fever be high, there is an insatiable thirst, while the secretions all over the body, urinary and perspiratory, are during the period of excitation markedly decreased. If the former be examined there will be found the ordinary accompaniments of pyrexia including a variable, but generally small, amount of albumin.

After these symptoms have continued for a few days, usually before the end of a week, the symptoms of irritation gradually diminish. Less complaint is made of the headaches, the distressing shrieks and outward manifestations of agony cease, the twitchings, spasms, and contractures become lessened and relaxed, the evidence of vasomotor spasm is followed by that of beginning vasomotor paresis, the pulse loses its rigidity, and blood pressure falls. The patient is quiet, the frenzy and the delirium take on a more subdued complexion, the disease passes from the stage of excitation into the stage of depression. Only the temperature remains elevated.

From this time onwards most of the motor symptoms, which were before characterized by irritation manifestations, now assume a paralytic character. The rigidity of the neck becomes less pronounced, and attempts to bend the head and move the neck call forth no remonstrance from the patient. Vomiting becomes less severe or ceases entirely. The abdominal muscles are no longer rigid and contracted, and frequently distention of the intestines, on account of loss of their

tonicity, causes pronounced tympanites. There is a continual overflow dribbling of urine if the bladder is left to care for itself, and, as the patient passes more deeply into a comatose condition, the obstinate constipation which was such a pronounced symptom in the beginning is succeeded by involuntary evacuations.

The pulse loses its resistant, small quality; the rapidity remains, as in the beginning, from 100 to 160, and the rhythm is disturbed. Sometimes when the patient is lying very quietly in a state of profound depression of consciousness, the pulse rate drops very materially, even as low as 60 or 70, but on slight excitement and postural change it becomes very rapid. The respirations as the patient passes into the stage of depression lose their excited, jerky character, and become very much slowed, with a tendency to interruption in the middle of an inspiration. This character increases and passes into typical, "grouped" respirations of the Cheyne-Stokes type.

In this stage the pupils are widely dilated, particularly if the stage is well advanced, and one pupil may be larger than the other. The pupils do not respond to any form of retinal stimulation. If the optic disc be examined, profound choked disc or optic neuritis is found. Strabismus and other ocular deviations in the second stage of non-tuberculous leptomeningitis are rare phenomena unless the exudation be profoundly marked at the base, but ptosis of one side is sometimes present. The countenance loses its pinched expression, and frequently the face as well as the surface of the body is bathed in clammy perspiration. Oftentimes there is considerable difficulty in swallowing, associated with other bulbar symptoms, all of bad omen. In a day or two after the symptoms of excitation have ceased the patient does not have the slightest cognizance of his surroundings, pays no heed to what is said to him, makes no outward manifestations of suffering, and, although the delirium continues, its character becomes profoundly altered. Instead of the active state of exaltation there is a low, unintelligible production of sounds and muttering, constituting the condition often described as typhoid. The temperature, as before mentioned, sometimes keeps up almost throughout this entire period, and not infrequently a short time before death, and particularly if the patient be alcoholic, the degree of hyperpyrexia which exists with all the outward manifestations of apyrexia, such as cold skin, extremities, etc., is astonishing. Sometimes it reaches 107°–109°. It is not, however, the rare exception for the temperature to fall during this stage until it is one or two degrees subnormal.

More or less marked paralytic manifestations generally make their appearance with the beginning of the stage of coma. These may be of an extremity, sometimes of one side of the body alone, sometimes

transitory, at others remaining until death or after recovery. Naturally they may be of any part of the body, depending upon the amount, intensity, and seat of the exudation. If the severity of the inflammation has been expended on the convexities, and if it be associated, as it very frequently is, with an inflammation of the superficies of the cortex, the extremities of one or both sides of the body will be implicated. It is believed that the extensors of the extremities are more liable to be palsied than the flexors. The paralysis has the characters of a flaccid palsy, myotatic irritability is lost, and there is no response in the part when subject to tickling or pricking. If the exudate be well marked at the base of the brain, it may surround and compress the cranial nerves from the second to the seventh, and the result is corresponding perversion of function.

The appearance of symptoms indicative of paralysis of nerves of bulbar origin, which is sometimes seen after the disease is far advanced, is a common forerunner of speedy dissolution. With them the clinical manifestations are unmistakable; the progressive impairment of respiration, of circulation, and of swallowing is attended with a complexity of symptoms most distressing even to observe.

Occasionally purulent meningitis, and of a severe form, occurs without being attended by any pronounced symptoms. Oftentimes the mildness of the symptoms is very misleading to the physician. I have recently seen a child, thirteen years old, convalescent from a mild attack of varicella develop such symptoms as restlessness, irritability, agitation, diarrhœa, irregular rise of temperaure, at one time 99°, at another time, a few hours later, 103° or 105°, deviation of the ocular axis, intermittent and slight, and stiffness of the neck which was to be made out only by careful examination. After these had extended over a period of ten days or longer, there gradually developed a condition of stuporousness in which the patient lay for hours and days at a time without moving, the only symptom that could be made out being inability to close the left eye, optic neuritis, and, because the extremities of one side of the body were never seen to move, it was suspected that there was a degree of hemiplegia. The child died apparently from heart failure, and on post-mortem examination there was found a most extensive purulent meningitis of the convexities and of the base which extended throughout the pia of the cord as far as the inferior termination. Clinically there were none of the acute symptoms, such as headache, vomiting, constipation, and erethism, that are so common in the beginning of a typical case of coccal meningitis.

Etiology.

The etiology of meningitis is emerging from the obscurity in which until the past decade it has been enshrouded. In fact, as has been hinted in the introduction, it is in the causation and diagnosis of meningitis that material advance has been made. The progress of bacteriology is responsible for the former; the knowledge that the intermeningeal space can be tapped with impunity in the lumbar region, the other. One of the factors which is responsible for incomprehension of meningitis from an etiological and pathological standpoint is the slowness with which clinicians accepted the bacterial origin of most inflammatory conditions, the other is the obstinacy which was manifested against considering the pia the same as any other serous membrane, and liable to the same diseases.

As in the case of every other inflammatory disease, the causation of meningitis may be considered under two heads—first, the conditions which precede the attack with such invariability as to be considered predisposing, and second, the exciting causes.

The predisposing causes are a variable quantity in each case.

The disease is more common in early adult life than at either extreme, although we shall take occasion later on to say a word about acute true meningitis of the new-born and the pseudomeningitis of the senile. It occurs more often in males than in females, this predilection being explained by the greater liability of the male sex to habits and experiences, such as alcohol and injuries, which are contributory factors to the disease. Leptomeningitis is more common, in every form, so-called idiopathic, sporadic, or epidemic, in the spring and in the winter than during the other seasons, and this not alone because of certain climatic conditions existing at these times which are favorable to bacterial development, but because the infectious diseases are of more common occurrence in winter. Of the habits to which mankind is addicted, the most common for the production of meningitis is excessive indulgence in alcohol, and this is readily understood when it is kept in mind that a disordered condition of circulation of the brain and membranes is a physiological effect of alcohol.

All those conditions which predispose to acute inflammatory diseases, such as exposure to wet and cold, particularly if the tone of the patient is in a depraved state from overwork, from worry, anxiety, or anterior disease, prolonged and exhausting application of the mind, exposure to excessive temperatures or the direct rays of the sun, or to atmospheres that are noxious and enervating, slight or

repeated trauma to the head, the presence of another bodily disease, all predispose to meningitis. Some writers believe that there is slight predilection to the development of meningitis in a neuropathic disposition, but this is in opposition to the impression that those of a neuropathic disposition are not so liable to inflammations of serous membranes.

The actively exciting causes may be classified as follows:

I. Traumatic and infectious.

II. Contiguous and infectious. Direct infection.

III. Infectious. Metastatic.

In a certain number of cases of meningitis the most diligent search fails to reveal any causation save trauma, which may have been so insignificant that one is loath to attach serious import to it. Such cases are apt to develop mild meningeal symptoms which terminate in recovery in a short time. A much larger number of cases, but still small when compared with the entire group, are those that follow severe injury to the head—injury that not only wounds the epicranial coverings but the skull as well. With these injuries are reckoned those inflicted by operation on any part of the cephalic extremity, the cranium, the nose, the eyes, the ears, or the face. Meningitis developing after operation is in these days of wholesome and fastidious surgery comparatively rare. The wounds that are most apt to be complicated by meningitis are the penetrating and crushing wounds, the first on account of the ease with which they carry infection to the membranes, and the second because of the difficulty which they offer to cleanliness. Penetrating wounds of the eyeball, with or without cyclitis, are very prone to excite meningitis. A striking example of this kind under personal observation was that of a man who in a street brawl had the ferrule of an umbrella pushed into his eye. He was taken to the hospital and the eye treated in the most approved way surgically, and, save for some appearances of alcoholic delirium, the patient made satisfactory progress. Yet three weeks later a chill ushered in uncontrollable headache, which was soon followed by most severe meningeal manifestations, to which the patient succumbed during the second week. At the autopsy the inner wall of the orbit was uninjured, but the parietal and visceral pia over the convexity of the corresponding hemisphere was the seat of a purulent exudate, which was prolonged through the tela choroidea into the lateral ventricles. It is, we believe, entirely within the bounds of truth to say that in every case of traumatic meningitis that comes to autopsy there will be found some bacterial organism to explain the occurrence of the disease.

Under the second category are included those cases that develop

from and in connection with pyogenic disease of adjacent structures and cavities, such as the mastoid and middle ear, the cavities of the nose and antrum of Highmore, the sphenoidal and ethmoidal sinuses, and the sinuses of the frontal bone, the cells of the ethmoid and the sphenoidal fossa, septic disease of the orbit or its contents, acute inflammatory conditions of the bones of the skull, such as osteomyelitis (a very rare condition), and septic diseases of the coverings and structure of the cephalic extremity. Of the latter may be mentioned, in the order of their importance, erysipelatous inflammation of the deeply seated structures at the junction of the jaws, the skull, and the neck, anthrax, carbuncle, furuncle, suppurative parotiditis, purulent inflammation of the tonsil (Beck), angina Ludovici, and, in short, any condition excited in the soft or hard parts by a specific bacterium. Of the causes included in the second category those attributable to the mastoid process and ear, particularly the middle ear, are most important. A purulent otitis media that has existed without special symptoms, with periods of exacerbation and apparent intermissions, for many years, may suddenly under some undiscoverable influence light up an attack of meningitis with or without infective thrombosis of a sinus, preferably the sigmoid; or the affection of the pia may follow burrowing of the purulent matter which leads to rupture of the tegmen tympani, and be associated with extradural abscess just over the tegmen. Leptomeningitis may follow operation on the middle ear such as for the removal of polypoid masses from the tympanic cavity, particularly if the removal be attempted through the external ear, for it is next to impossible to render completely aseptic the tympanic cavity and antrum, even when the latter have been approached from behind the ear. It may develop apparently directly from such granulation masses themselves.

When an attack of meningitis is traceable to purulent otitis media, there is almost always a history of cessation of the aural discharge for some days previous to the meningeal symptoms, the same as in abscess of the brain traceable to a similar cause. The mode of infection in these cases is most frequently by means of an infective sinus thrombosis, but in some cases, as when the petrous bone is itself the seat of extensive pathogenic process, the infective inflammation passes along the sheaths of the seventh and eighth nerves. Next in causative importance to disease of the middle ear and to pyogenic process in the petrous portion, come mastoiditis, furuncle and carbuncle, and diseases of the external ear.

Leptomeningitis may result directly from phlegmonous inflammation of the nose, the veins of the nasal cavity being the pathway of infection, as they are in those cases in which the inflammation of the

pia follows operation on the nose for the removal of polypi (Quinlan) and for the cure of hypertrophic rhinitis (Wagner). When involvement of the pia follows purulent disease of the ethmoid cells the infection occurs through the lamina cribrosa. Infection of the sphenoidal fossa is most frequently complicated by thrombosis of the cavernous sinus, but next in frequency is a basal meningitis which eventually extends to the convexities; in these cases it is probable that the infection sometimes takes place through the bone diploë. Purulent disease of the frontal sinuses predisposes to brain abscess much more than to meningitis; in fact, the latter is an extremely rare complication.

Orbital abscess, cyclitis, purulent inflammations of all kinds have been followed by meningitis, complicated or not by brain abscess.

Under the third heading, the infectious, there are included all of the infectious diseases, and especially those that have been proven to be dependent upon a specific organism. These, named in the order of their importance as causes of meningitis, are pneumonia, typhoid fever, cholera, dysentery, influenza, remittent fever, and gonorrhœa. Meningitis occurs sequentially to scarlatina, to measles, to variola, to varioloid, and to varicella, but in these cases it is probable that most, if not all, have been preceded by an otitis media purulenta, the direct consequence of the acute disease, or are associated with pneumonia.

Ghika, Sabrazes, Mills, Comibas, Neumann and Schäffer, Bozzolo, and many others have brought forward positive evidence of the all-important influence of the pneumococcus in causing meningitis.

As we become more familiar with the diplococcus pneumoniæ we recognize how widely distributed throughout the body it oftentimes is. Its presence in the meninges was shown soon after its isolation by Fränkel, Foà, Bordoni-Uffreduzzi, Weichselbaum, Ortmann, and others, but it was, as Guinon has shown, the striking paper of Netter that first drew attention to the etiological significance of these findings. In each succeeding year during the past decade contributions have appeared which demonstrate the extremely important place which this bacterium has in producing meningitis. This coccus is found in the meninges with such frequency relative to the lungs, that Foà has proposed to distinguish it according to its principal locality, as the pneumococcus and the meningococcus. Its presence in the meninges gives rise to an exudative inflammation whose product may be predominantly serofibrinous, seropurulent, or fibrinopurulent, and this most often without simultaneous disease due to the coccus in other parts of the body, such as pneumonia, inflammation of the endocardium, etc. The avenues of entrance for the cocci are in all

probability, for the great proportion of cases, the nose and mouth, and next most frequently through the maxillary and tympanic cavities and the cribriform labyrinth to the meninges by way of their vascular and lymphoid intercranial communications. Hubbenet believes that the cocci from a focus of pneumonia may pass through the loose connective tissue of the mediastinum between the œsophagus, the cervical vertebræ, the trachea, and the carotid, and so to the pia. Cultures of the cocci made from the meningeal exudate injected within the cranium of animals causes meningitis of a purulent character, as will also cultures of the pneumococcus taken from the lungs.

Meningitis due to this bacterium may occur independently of pneumonia, or it may precede, occur coincidently with, or follow it. In pneumonia, and, in short, in any other disease due to the diplococcus pneumoniæ of Weichselbaum, the cocci are widely distributed throughout the body, and it depends upon the resistance of such individual parts as are known to be prone to infection whether or not they will escape.

That leptomeningitis sometimes occurs with and following typhoid fever is a fact borne out by statistics; but that this association is an extremely uncommon one is shown by the results of two thousand autopsies from the Munich Pathological Institute, in which it was found only eleven times.

In the exudate of cases of meningitis there have been found not only the bacillus coli communis but the specific bacillus of typhoid fever, the latter without the presence of intestinal lesion. In two cases recently reported by Tietine, one of acute serous leptomeningitis, the microscope revealed the presence of typhoid bacilli in the pia, and cultures showed them also. In the second case, in which the exudate was purulent, a similar condition was found, and inoculation of animals with the cultures gave rise to a seropurulent meningitis; so that it would seem probable that the typhoid bacillus may exercise an injurious effect on other parts of the body than the solitary glands of the small intestines, and that affection of the latter is not absolutely necessary after systemic infection.

In a certain number of cases of typhoid fever there are meningeal symptoms, in which after death there are found no pathological conditions of the meninges to account for them. These cases are really examples of spurious meningitis or pseudomeningitis.

A study of the mortuary list of the great cities in this country during and after the epidemics of influenza which prevailed here with great severity in 1891, and to a lesser extent in 1893 and 1894, showed a marked increase in the number of deaths attributed to meningitis. Clinically the superimposition of meningeal irritation symptoms dur-

ing an attack of influenza has frequently been noticed; so frequently, in fact, that by some it has been thought fitting to refer to a meningeal form of influenza. As we shall point out later in discussing symptoms, the types of meningitis supposedly due to influenza are of the mildest kind, and are rarely, if ever, fatal; therefore no serious attempt has been made to discover the bacillus, even in cases produced artificially in animals.

In addition to these there are other cases of meningitis which are produced by the two pyogenic organisms, streptococci and staphylococci. Just as in a certain proportion of the cases of pneumonia the only organisms to be found are the two cocci just mentioned, so it is in leptomeningitis. It is through the direct action of these cocci that leptomeningitis arises in such conditions as ulcerative endocarditis, in general septic conditions such as puerperal septicæmia, etc., as well as in localized septic conditions such as purulent otitis media.

That leptomeningitis of an acute purulent character may be due to the gonococcus, would seem to be proven. Stenon has published a case which was apparently due to the gonococcus. Other cases attributed to this cause have been mentioned by Hayem and Parmentier, Stanley, Gull, Chavrier, Teissier, etc. The liability of this coccus to produce peritonitis, monoarthritis, and polyarthritis is well known, and from analogy it would seem that meningitis might be due to it.

Epidemic cerebrospinal meningitis has been in the past, and is yet by many, considered a disease apart from acute leptomeningitis, and insomuch as it is caused by a specific organism, and has a clinical history and course characteristic of infectious diseases, it deserves to be. It is yet no whit the less a form of meningitis, just the same as any other form of coccal meningitis is. In epidemic leptomeningitis bacteriological investigation has shown that this disease is dependent upon constant bacterial factors, and that certain bacteria are present with absolute constancy, possibly one for the sporadic, another for the epidemic, form. The disease is apparently becoming more prevalent, but this may be due to its more common recognition and differentiation. Within a few years epidemics have prevailed in this country, and very careful study of the disease has been made by J. Lewis Smith, and by Flexner and Barker, who carefully and scientifically studied the epidemic in Lonaconing, Md.; and although no genuine epidemic has recently occurred in Europe, small epidemics have been reported by Monk, in Shropshire; by Lemoine, as it occurred in two companies of French artillery; and by Wollf who reported one hundred and thirty-two cases which occurred in six years in the Hamburg Hospital. The investigations made by

Foà and Bordoni-Uffreduzzi of the Turin epidemic in 1885 and 1886, and by Leichtenstern of the Cologne epidemic, have been of great importance in placing this disease in its proper etiological category.

Just as pneumonia may prevail epidemically, just as it may seem for years to have certain centres or foci of occurrence, so may epidemic meningitis. Like other infectious diseases, its place in the mortuary lists of great cities is never vacant, and like them it is liable, occasionally, to devastate sections of the country under the influence of certain antihygienic conditions, particularly those of defective sanitation and water-supply. In a general way, the etiological conditions that are true for acute purulent leptomeningitis are true as well for the epidemic form, except that in the latter children fall easy victims to the disease.

The pathway of infection in the great majority of cases of epidemic meningitis is through the nasal and buccal cavities, as well as through the lymph system. The real excitant of the epidemic form of the disease is a coccus. Weichselbaum described it in 1887 under the name of diplococcus intracellularis meningitidis. By many bacteriologists it is considered a variety of the pneumococcus, by others an organism quite different. One of the most recent investigators, Jaeger, has probably settled the question. He believes that the diplococcus intracellularis is not identical with the diplococcus pneumoniæ of Fraenkel-Weichselbaum, but that the former is the cause of the cases that occur epidemically and the latter of sporadic cases. The diplococcus intracellularis which he proposes to call the tetracoccus intracellularis is constant in the nasal secretions and is possessed of the property of retaining its virulence for a long time in a dried state.

Pathology and Bacteriology.

In the present state of our knowledge of inflammation of the meninges it is necessary to admit and describe a meningitis in which the pathological product is predominantly a serous or serofibrinous exudate with a few leucocytes and occasionally a pus corpuscle. These cases sometimes constitute examples of acute hydrocephalus. The pia, predisposed to diseases from inherent or acquired causes, becomes inflamed by the action of non-pyogenic organisms, which cause a simple exudation that is poured out on the surface of the visceral pia and collects in the intermeningeal or subarachnoid space. The accumulation of this exudation may cause so-called external hydrocephalus. This accumulation of fluid in the subarachnoid space

disturbs the reciprocal pressure relationship existing between the extraventricular and intraventricular spaces, and an accumulation of fluid with accompanying distention of the ventricles, acute hydrocephalus, is the consequence. This collection of extraventricular and intraventricular lymph exercises a malign influence on the convolutions, and if it be not of short duration leaves a permanent impress in the shape of disordered functions. These conditions are considered in greater detail under hydrocephalus.

In acute leptomeningitis of the customary form—that is, the form in which pus in greater or lesser quantities is one of the pathological products—the extent of the lesion, circumscribed or diffuse, depends upon the cause and the intensity of the disease.

In those instances in which the involvement of the meninges has been secondary to adjacent localized purulent foci, as otitis media purulenta, orbital phlegmon, and the like, or in which the infective process has followed injury to the cranial bones, the intensity of the inflammation and consequent collection of pathological products will be considerably localized, generally on the lateral or vertical surface of one hemisphere. The fact that acute leptomeningitis extends over the entire surface of the central nervous system, and by means of the tela choroidea into the interior as well, with great rapidity, is often impressed on one at the autopsy of these cases.

The exudation in the purulent forms of acute leptomeningitis varies in its physical characteristics according to the bacterium which causes it. Often the exudation is principally serous in which large amounts of pus corpuscles and leucocytes, with a small quantity of flaky matter and fibrin, are found. As the inflammatory process is predominantly in the parietal pia, and secondarily in the visceral, the serous fluid naturally distends the intermeningeal space, while exudation of leucocytes occurs into the perivascular spaces and along the coats of the blood-vessels. The transformation of the latter into pus which ensheaths the vessels and sinks into and fills up the fissures, gives a striking appearance when the dura is opened. If the leptomeningitis is diffuse and involves the base of the brain, the entire spinal axis may be ensheathed in a seropurulent exudate extending even to the bottom of the spinal canal. At such places as the junction of the oblongata and the cerebellum, where the bridging of the parietal pia forms the oblongata-cerebellum cistern, the collection of seropurulent exudate and the consequent matting together of the blood-vessels and nerves at the base of the brain are the greatest. The coats of the blood-vessels are infiltrated and studded with leucocytes, and the veins are often plugged with coagula. The seropurulent exudation and infiltration are not confined to the intrameningeal

space and meshes of the pia, but by means of the purulent infiltrated tela choroidea pass through the substance of the brain into the ventricles, and the consequence is that the choroid plexus is found to be swollen and covered with pus and seropurulent exudate, the contents of the ventricles are increased, flocculent, and turbid, and the ependyma has a dull, scaly appearance.

The common pathological accompaniments of acute purulent leptomeningitis are changes in the dura and in the cortex of the brain. On removal of the calvarium, the veins of the diploë are congested, the venous sinuses are distended, and the blood-vessels between the layers of the dura are congested, the seat of passive hyperæmia and distention. When the dura is incised and stripped back there are oftentimes seen small areas of pachymeningitis interna. The parietal layer of the pia, robbed somewhat of its glistening appearance, is still transparent and allows the congested, distended vessels of the visceral pia and the greenish-yellow purulent and seropurulent exudate which is collected into patches or diffuse, filling up the fissures and tumefying the vessels and pia, to shine through.

The changes in the brain, in the gray matter of the cortex, very rarely in the white, are directly in proportion to the intensity of the inflammation and the duration and severity of the exudate. It is readily understood why such changes occur if it be kept in mind that prolongations from the adventitial sheaths of blood-vessels encapsulate each cell, thus giving it a perivascular capsule. The lymph spaces that are thus formed are continuous with the pial space. It is probable that in every case of infective origin some change takes place in the cortex. Until lately these have not been recognized because of inadequate methods of staining, a procedure which is absolutely necessary to enable us to detect the changes. The Nissl method of employing methyl blue, which stains the ganglion cells and their prolongations and reveals the condition of the vessels as well as that of the cellular proliferations, is nearly an ideal one for the purpose. In a case of meningitis on which I recently made an autopsy I was able to make out most distinct changes in the cortical blood-vessels and the cells of the small and large pyramids by the use of this method. The changes in the cortical tissue, which are mediated principally by the extension of inflammation along the pial sheaths of the veins, or by direct extension of inflammation following on adhesion of the pia to the cortex, are minute extravasations in the perivascular sheaths and distention of the perivascular spaces and subsequent encroachment upon the nerve cells. These may be very numerous, and, depending upon them and their severity, will be the changes which can be detected in the ganglion cell. The blood-ves-

sels themselves are overfilled and tortuous, and infiltration of the walls and the surrounding tissue is strikingly manifest.

These conditions in the cortex are sometimes evident to the naked eye, but there are frequently slight punctate spots to be seen in the cortex, which in connection with hyperæmia of the vessels and the tumefaction of the brain tissue give an easily recognizable appearance, a condition which if it were a little farther advanced would deserve the name red softening, such as is described in the article on diseases of the brain under hemorrhagic encephalitis and abscess of the brain.

In rare instances the changes in the brain tissue go on to the formation of abscess, especially when the meningitis is secondary to trauma, to disease of the middle ear, or to sinus thrombosis. When it does, the purulent condition in the brain is a very diffuse one and the symptoms of abscess may be obscured. Bacteriological examination of the meninges and of the exudate after death, and sometimes of the exudate obtained by means of lumbar puncture of the subarachnoid space, reveals the presence of specific organisms. As we have pointed out, some of these are found with such constancy that they are now considered provocative of the disease.

The most commonly found organism is the micrococcus pneumoniæ crouposæ, the diplococcus pneumoniæ of Weichselbaum and Fraenkel, a spherical, oval, or lancet-shaped cell, generally seen in double cocci or in a spherically shaped chain, very rarely in spread-out colonies. Some of these diplococci are naked, while others are covered with a gelatinous capsule; but covered or uncovered alike, they stain with fuchsin and gentian violet dissolved in aniline water, and those stained with the latter when treated with a solution of iodine and alcohol do not part with the stain. Cultures of them are made on the ordinary media, except gelatin, with great readiness, at a temperature varying from 22° to 39° C. Although the activity of this diplococcus, the diplococcus lanceolatus or meningococcus, does not cause a really typical inflammatory exudation, the exudate attending meningitis caused by this bacterium is somewhat characteristic. It is of a cream-yellow color, very rarely greenish or tinged with blood, of a viscid, slightly tenacious consistence, with a tendency to smear. The degree of purulence may vary from serofibrinous exudate with a small number of pus corpuscles detectible only with the microscope, through fibrinopurulent to a clear purulent. It occurs in the meshes and on the surface of the pia, on the surface of the brain and in the sulci, particularly at the base. In 25 cases of suppurative meningitis under personal observation analyzed by Netter, there were found in 18 the pneumococcus, in 4 the streptococcus pyogenes, in 2 the intracellular

diplococcus of Weichselbaum, in 1 the typhoid bacillus, in another the pneumococcus of Weichselbaum, and in another certain unknown bacilli. In 45 cases from the literature there were found, in 27 the pneumococcus, in 6 the streptococcus, and in 10 the intracellular bacillus of Weichselbaum.

Possibly this bacillus and certainly the diplococcus intracellularis are the bacteria that are responsible for the form of leptomeningitis described as epidemic cerebrospinal meningitis. In these cases the purulent exudate is found with a corresponding degree of severity in the pia of the cord, particularly in its cervical portion, extending often to the cauda equina and being most severe on the posterior surface.

It cannot be said positively until the bacteriology of influenza is known whether the specific organism of that disease causes the meningitis which sometimes accompanies influenza or whether the latter is due to the bacteria which we have been describing. The type of influenza in which cerebrospinal symptoms are the predominating ones simulates very closely the mild form of epidemic cerebrospinal meningitis, and it is more than probable that they are caused by similar organisms. It is so very infrequently, however, that the influenza form of meningitis terminates fatally, it being, as has before been said, the mildest of all forms, that we cannot speak dogmatically on this point. It is not improbable that many of the cases of influenza which develop "meningeal" symptoms, such as those described by Quincke, are cases of meningitis serosa, a pathological condition which I prefer to call pseudomeningitis.

The next most common bacterium which is found in the exudation of leptomeningitis is the streptococcus. It is found with and apart from the diplococcus pneumoniæ, it admits of ready cultivation, and when inoculated into the meninges of animals it produces a purulent inflammation. In a few instances it has been associated in the exudate with the bacillus coli communis and the staphylococcus, but these latter two, as well as the gonococcus, which has been found by some authors, have not been found alone in the exudate, and the part they play in producing the inflammation is not established with any degree of certainty.

A number of bacilli of meningitis have been described, some of them, it has been thought by their discoverers, of sufficiently specific characters to be called the bacillus of meningitis. We have already mentioned that Foà gave this designation to the diplococcus of pneumonia when it is present in the meninges. The coccus described by Weichselbaum, the diplococcus intracellularis meningitidis, has already been considered in its relationship to epidemic cerebrospinal meningitis. We have spoken of the bacillus which was isolated by

Neumann and Schaeffer, which they thought might be the bacillus of meningitis and which had very striking resemblances to the bacillus of Eberth, but like the discoveries of others their findings have not been corroborated.

The kind of bacillus that most often causes purulent meningitis, next to the tubercle bacillus, is the bacillus of typhoid fever, the bacillus of Eberth. The first of such cases to be reported were, as has already been mentioned, those of Roux and of Adenot; but in both of these cases there was some doubt about the identity of the bacillus described with the typhoid bacillus. The findings by Kamen and of Honl put the matter beyond cavil. Since their contributions, cases in which the typhoid bacillus has been found in the meningeal exudate have been reported by Hintze, Fernet, Monsi and Carbone, Stühlen, Tietine, and others.

The next most common bacillus is the bacillus coli communis. This has been found in the exudate by Howard, Biggs, Sestre and Gaston and others, and very recently by Scherer.

The bacillus pyocyaneus has also been claimed as the causative agency by Kossel and by Pesina and Honl.

Of the pleomorphic bacteria that have been found in the exudates of meningitis we can here mention the cladothrix asteroides of Eppinger, and the actinomyces described by Moosbrugger, by Honl, and by Lezine.

Epidemic Cerebrospinal Meningitis.

Epidemic cerebrospinal meningitis is an acute infectious disease. It is, however, a form of genuine meningitis, and as such mention of it must be made here. The various organisms which have been claimed to be the real cause of this form of meningitis have been described above. Here I desire to express the belief that epidemic cerebrospinal meningitis is due to the presence in the cerebrospinal meninges of the diplococcus intracellularis of Weichselbaum, or, as Jaeger proposes to call it, tetracoccus intracellularis, and which is frequently referred to as the meningococcus intracellularis to differentiate it from other micro-organisms found in meningitis, especially the pneumococcus. The infection is probably mediated through the lymph spaces, the corporeal source of the infection being the mucous membrane of the nose and the mouth. I am not unmindful that there has been a considerable amount of evidence brought forward to prove that the pneumococcus is the organism which produces this disease, and that Ormerod teaches that the pneumococcus is the *sine qua non* of its occurrence. After careful sifting and weighing of the real evidence at hand the conclusion is forced upon me that when

cerebrospinal meningitis prevails epidemically the diplococcus intracellularis will be found in the purulent exudate, and that cultures inoculable to susceptible animals can be made from this exudate. When the disease occurs sporadically, I believe further that the diplococcus intracellularis is not present, but one of the common cocci of purulent meningitis, and especially the pneumococcus, is present. Naturally the diplococcus of pneumonia is found in cases of epidemic cerebrospinal meningitis, and it unquestionably has been demonstrated by several bacteriologists; but these bacteria are commonly found in cases of purulent meningitis, not epidemic, in which the intracellular diplococci are absent. It must also be said that in many of the contributions to the bacteriology of meningitis accurate discrimination has not been made between ordinary cerebrospinal meningitis and the epidemic form. It seems to me impossible for any one to study the bacteriological literature that has grown up about the various forms of purulent meningitis during the last quarter of the present century, and particularly the controversy of some Italian investigators, without coming to this conclusion. The *pros* and *cons* of the question are discussed in another connection, but attention must be called here to the strictly scientific confirmatory evidence that has recently been contributed to the support of the above contention by Heubner, who tapped the subarachnoid space in the lumbar region, and from the fluid thus obtained made cultures of the meningococcus intracellularis with which susceptible animals were inoculated.

Epidemic cerebrospinal meningitis has a particular interest for American physicians insomuch as the disease has prevailed more extensively in this country than in any other part of the civilized world. Treatises on epidemiology and works such as Hirsch on the geographical and historical pathology of this disease describe three very distinct epidemics occurring in this country and in Europe. The first of which we have accurate description extended over the entire first quarter of the nineteenth century, with temporal intermissions of one or more years, and geographically spread over the New England States and those of the Middle Atlantic seaboard, reaching inland as far apparently as any considerable civilization had progressed. Then for about fifteen years there was a cessation of activity, and the disease next broke out in the West and South in about 1840. Limited epidemics occurred off and on until 1846. The third great epidemic in this country started ten years later, reached its height during the Civil War, and did not subside until ten years after the war was at an end. In this epidemic the geographical distribution was very irregular, the disease prevailing at one time in New York, at another

in the Carolinas, and again in Philadelphia. A study of the medical history of the War of the Rebellion does not throw much light on the disease in reference to its prevalence under certain conditions, the most valuable sources of information in this country being the writings of Stillé and Smith.

During the last decade of the present century the disease has existed in this country almost continuously, but very rarely prevailing to such an extent as to call any considerable attention to it. The epidemic of most importance of recent date is the one of Lonaconing, Md., in 1893, which was studied by Flexner and Barker. During the severe epidemic of influenza in 1891 many of the deaths attributed to influenza were thought by some to be really due to this disease.

Clinically the disease occurs in very many forms, but usually two distinct types, the mild and the severe, are described. And although a recitation of the symptoms of these two varieties may be made to include all the manifestations of every form of the disease, it is necessary to say in the beginning that no two cases during an epidemic will be counterparts. Some cases run an abortive course, others an intermittent and irregular one, but they all have certain distinct symptoms in common. Frequently the severe or foudroyant form is common at the beginning of an epidemic, and the mild or abortive type towards the end.

The symptoms which characterize the severe form are the sudden onset of the irritation symptoms and their great severity, which may or may not be preceded by a profound chill. A feeling of depression and portentous dread may be present for a time before the chill, then follow a sharp rise of temperature, intense and agonizing headache and backache, associated with rigidity of the neck and back, alone or with such universal tonic condition as to produce the most violent opisthotonos or pleurosthotonos, palsies of some of the ocular muscles, violent delirium, rapidly deepening coma, and death, which may occur before the end of the second day.

In the less violent and usual form, the victim after a day or two of malaise, indisposition, and chilly feelings, begins to complain of dizziness, vertigo, hypersensitiveness to light, noise, and all forms of mental excitation, associated with a feeling of increasing depression. The pain in the head and neck becomes more severe, of a boring, intolerable character, and radiates to the lumbar region. As the vertigo increases, uncontrollable vomiting, the act of which gives no relief, adds to the patient's suffering. Frequently the patient, before delirium obscures the intelligence, complains piteously of pain when any part of the body is touched or when he is moved. The temperature ranges from 100° to 104° F., the latter degree being rarely

reached, for it is not a disease of high temperature, nor does the temperature range bear a constant relationship to the severity of the disease. The pulse is accelerated, of small volume, and often during the paroxysms of pain, even in the beginning of the disease, is irregular. Rigidity of the neck and spasmodic contractures of a limb or of the domain of a cranial motor nerve are common early symptoms. The skin is almost always the seat of some eruption, which makes its appearance even on the first days of the disease. This may consist of simple erythematous or urticarial eruption, but some form of herpes, particularly of the lips, or that occurring symmetrically on the extremities, is the most common eruption. The cutaneous manifestations, like many of the other symptoms, vary with the epidemic; in some epidemics they are the rule, in others the exception. The frequency of occurrence of petechial eruption in this disease led some of the older writers to refer to it as "spotted fever," an unfortunate and useless designation.

The sphincters are involved very early in the course of the disease, and incontinence of urine in the beginning is quickly followed by retention, a condition which should be attended to with the catheter. When the stage of coma sets in, the symptoms do not differ from those of ordinary severe leptomeningitis.

In the mild or the so-called abortive type the symptoms may be mild from the beginning or they may set in with considerable severity, and then begin to mitigate before the delirium has become very pronounced. Only in very exceptional instances has recovery followed in a case that has gone on to coma. Towards the end of an epidemic there are always a number of cases in which symptoms of depression and slight meningeal irritation only are present, such as vertigo, headache, pain and soreness in the back, hyperæsthesia, and somnolency, just as mild cases occur at the wane of almost any other epidemic. These are by some considered as very mild cases of the disease and by others as forms of pseudomeningitis, due perhaps to some toxic substance which causes a serous exudate, but not to the diplococcus intracellularis, the essential causation of the genuine epidemic form. It would seem probable, however, that they are due to the same organisms, but occurring under conditions which have deprived them of virulency.

An impressive fact that we have learned from recent epidemics in this country is that the occurrence of epidemic cerebrospinal meningitis is governed by the same features, if we may not say the same laws, as are all other epidemics and that a better knowledge of epidemiology will eventually eradicate the disease, as it has nearly eradicated typhus fever and made influenza less terrible.

In many of the less severe forms of epidemic cerebrospinal meningitis, life is spared, but at the expense of some frightful deformity, such as paralysis of a limb or of one side, of blindness, deafness or mutism, of imbecility, etc. In other cases a chronic process of inflammation of the meninges or hydrocephalus is left behind, which eventually becomes the contributing cause or the direct cause of death. In 64 cases that convalesced after a Heidelberg epidemic, Moos found deaf-mutism in 38, absolute deafness in 20, partial deafness in 1, and a staggering gait in 32. In the Lonaconing epidemic Randolph found that four-fifths of the cases showed some intraocular change, the most common being congestion of the retinal veins and optic discs, and not rarely optic atrophy.

The condition of the blood has been carefully studied by Flexner and Barker and by Rieder. The former found an increase of small, mononuclear round cells, lymphocytes; leucocytosis, the leucocytes showing oftentimes striking vacuolation; and large epithelioid cells with vesicular nuclei and some round cells.

The general symptoms and accompaniments of epidemic cerebrospinal meningitis are similar to those of other acute infectious diseases. The lips, teeth, and buccal mucous membrane are covered with sordes, the tongue is coated, the breath is often extremely foul, and occasionally there are profuse dysenteric stools, although constipation is the rule. The spleen is generally enlarged, the urine is very scanty, high-colored, and may contain albumin and blood. The most common coëxisting condition, one often seen in profound septic states, is a peri- and intra-articular effusion in the joints of the extremities, particularly the knee and the elbow. It has been noted by Flexner and Barker that the meningeal symptoms seemed to be favorably influenced by the advent of joint complications.

In addition to the cases from which leptomeningitis must be differentiated, and which will be considered under differential diagnosis, it should be borne in mind that the condition which is liable to be confounded with this disease, and from which in some instances it cannot be separated without a bacteriological and histological examination, is anterior poliomyelitis, a disease which no one will doubt at this day is of infectious origin and sometimes prevails epidemically. It was a distinction between these two diseases which was found so difficult to establish in the beginning of the epidemic which prevailed in Rutland, Vt., and vicinity, in the spring and summer of 1894, and which has been reported by Caverly. The same is to be said of the epidemic in Stockholm which was reported by Medin. If it be borne in mind that headache, delirium, and subsequent coma are the distinguishing features of meningitis, and that although

there may be pain, and that of great intensity, in the acute epidemic form of anterior poliomyelitis, it never assumes the features of that disease, nor does the paralysis of the former compare either in time and intensity with that of the latter.

Meningitis of the New-Born and of the Aged.

The first form of meningitis was seen more frequently before the days of aseptic midwifery, and is now, like tetanus neonatorum, a condition to which it is allied genetically, very rare. The source of infection in these cases was probably through the umbilicus, although other avenues cannot be denied. The disease is characterized by its abrupt onset, by the severity of the convulsive phenomena, and by the early fatal termination. At the autopsy a hemorrhagic, sanio-purulent inflammation is found. Clinically this form of disease is scarcely to be differentiated from the cerebral palsies of childhood, for one form of which it is responsible.

Nevertheless genuine cases of cerebrospinal meningitis occur during the first weeks of life which are in all respects similar to purulent leptomeningitis of adults. Rotch has reported a case occurring in an infant six days old. The symptoms were convulsions, frequently repeated up to fifty a day, and an abrupt rise of temperature. There was no retraction of the head, no opisthotonos, no ocular symptoms—in short, no symptoms except the two just mentioned. Examination of the exudate after death revealed the presence of the diplococcus pneumoniæ.

Meningitis of senility is in reality an infectious process, predisposed to by impoverished circulation attendant on marasmus of old age. It is of very rare occurrence. Clinically it is characterized by early appearance of delirium and rapidly occurring coma, which have not been preceded by prolonged manifestations of meningeal irritation, such as headache, stiffness of the neck, and vomiting, although the latter do occur. It invariably leads to a fatal termination.

False Meningitis; Pseudomeningitis.

1. *Meningitis Serosa.*—This is a form of meningeal affection which is not of bacterial origin and not associated with the presence of bacteria. The etiological factors that have been posited are injury to the head, persistent mental overstrain, acute and chronic alcoholism, acute febrile diseases, pregnancy, otitis media, influenza, and prolonged exposure to cold and wet. It is not a true inflammatory process.

Clinically the disease does not develop with the abruptness and severity of ordinary leptomeningitis, nor are any of the symptoms

save those of increased intracranial pressure, which depends upon the increase of extra- and intraventricular fluid, so intense. The symptoms, although they may be acute, generally develop insidiously, and particularly if the disease occurs secondary to conditions that produce hydræmia. Occasionally it develops after such injury as a fall from a chair, a blow on the head, and the like. In these cases the symptoms, consisting of headache, rigidity of the neck, vomiting, irregularly contracted pupils, come on soon after the injury and are followed by extreme dilatation of the pupils and often by double optic neuritis, spasms, delirium, and coma without the development of any considerable amount of fever (101°, 102°), or the fever may be entirely absent. After the occurrence of these symptoms, the increasing size of the head, if the disease occurs in patients before the fontanelles have closed and the sutures ossified, or signs of intracranial pressure if after this period, are the symptoms which follow these. In many cases this symptom complex develops with one of the acute diseases, such as gastroenteritis or typhoid fever, and then it becomes absolutely impossible without puncture of the subarachnoid space in the lumbar region, or of a ventricle through a fontanelle, to say whether the symptoms are due to a serous meningitis or whether they are dependent upon true leptomeningitis.

Not infrequently, after the affections has lasted for a few days, the intensity of the symptoms begins to abate, and the disease goes on to recovery. In other instances the clinical picture of hydrocephalus develops, which, if death does not take place soon, assumes a chronic form.

2. *Alcoholic pseudomeningitis* (wet-brain) is a condition similar to that just described, and consists of an œdematous state of the pia and brain substance found in patients who have been addicted to the excessive use of alcohol, and who die from the effects of it. It may occur with coexisting disease in other parts of the body such as pneumonia, complicating which it is often seen, or it may occur alone. In many of the cases of wet-brain that come to autopsy there is found a true purulent meningitis, but in these cases the infection has been superadded to the depraved state of the meninges the result of alcohol. The sources of such superadded infection, simple or mixed, are quite the same as in the ordinary purulent form.

The symptoms of alcoholic pseudomeningitis are those of serous meningitis plus those of alcoholism, acute or chronic, with their well-known symptoms of physical unrest and psychical excitation, and subsequent depression, followed by ever-deepening coma. Infection with the pyogenic organism is attended with an exacerbation of symptoms and the development of those of purulent meningitis; but these

are so masked by the enshrouding symptoms of alcoholism that, with the exception of the stiffness of the neck, they do not become apparent. The form of wet-brain which sometimes is seen complicating an attack of acute multiple neuritis, and which goes on to a fatal termination, I have seen attended with such a high degree of pyrexia (103°–107°) for two weeks' duration, unresponsive to all forms of antipyresis, save the Brandt method of tubbing; yet on autopsy the lungs merely showed stasis, no croupous pneumonia, while the intracranial findings were œdema of the meninges and cortex. Cases of "wet-brain" are said to be invariably fatal. Yet in one case, in which the coma and unconsciousness extended over a period of several days (nearly three weeks), and in which the diagnosis seemed to be clear, the patient recovered.

Diagnosis, Localizing Diagnosis, and Differential Diagnosis.

The diagnosis of acute leptomeningitis, it matters not what the pathological product is, is not such an easy matter as might be inferred from reading an account of the attending symptoms. When the symptoms are considered in connection with the causation of the attack, particularly the manner of their development after such causation, and when the headache, vomiting, nuchal rigidity, and hyperæsthesia of the entire nervous system develop, these symptoms having the characteristics which have been mentioned, the diagnosis is at once suggested. The occurrence of localized muscular twitching and muscle contracture, such as strabismus, twitching in the muscles innervated by the seventh nerve, rapid development of choked disc and optic neuritis, and more than all, paralytic manifestations developing particularly in places where irritation phenomena were manifested earlier, associated with progressively deepening coma, without other exacerbations than those of continual increase and without remission, are symptoms of the greatest suggestiveness in the diagnosis of the disease. Yet even these are not absolutely diagnostic and sometimes, if further aid cannot be invoked, only a probable diagnosis can be made.

The most important aid in contributing to the diagnosis of any form of meningitis, after a consideration of the symptoms and their manner of development, is the procedure known as lumbar puncture of the subarachnoid space—a procedure whose feasibility and utility have been shown particularly by Quincke, of Kiel, and the importance of which in facilitating the diagnosis has been insisted upon by Lichtheim and Jacoby. This procedure is based upon a knowledge of the fact that the subarachnoid spaces of the brain and spinal cord

are in direct communication, and that both may be emptied or injected at the inferior end of this space in the lumbar region. The trifling operation consists in passing a needle into this space between the third and fourth, or between the fourth and fifth lumbar vertebræ, the former height in adults, the latter in children, and about 5 mm. to one side of the median line. The size of the needle varies according to the age of the patient; in young children a needle of the diameter of a hypodermic needle and from 5 to 6 cm. in length answers the purpose, while for adults the needle should be at least 8 cm. long and 1½ mm. in diameter. The mistake most frequently made in first attempts at utilization of this procedure is that the needle is not carried to a sufficient depth. A needle with a stylet is to be preferred, particularly in adults. It seems to be the unanimous opinion of those who have employed this method that an aspirator should not be used. This operation is most easily performed in children when the patient lies on the abdomen across the knees of the nurse, the back being thus forcibly convexed in the lumbar region. In adults the patient should lie on the side if it is thought best to employ an anæsthetic, and this is often advisable, or in the sitting posture with the spinal column flexed on the pelvis if narcosis is considered unnecessary. As soon as the needle enters the subarachnoid space the fluid begins to flow, unless the lumen has become occluded by a piece of tissue or by coagulated blood. Microscopical and bacteriological examination of the withdrawn fluid, although it be only a few drops to show whether it be normal cerebrospinal fluid or altered in its chemical composition to suggest the presence of tumor, or changed in its physical characters by the presence of specific organisms, such as the diplococcus pneumoniæ, the diplococcus intracellularis, tubercle bacilli, streptococci, staphylocci, or any other specific organisms, is of paramount importance in making the diagnosis and the differential diagnosis. Cultures may then be made, and from these susceptible animals may be inoculated. In this way Heubner has recently been able to differentiate the epidemic form from simple purulent leptomeningitis.

Normally the cerebrospinal fluid contains a mere trace of albumin which can be detected only by use of the most delicate tests. In acute inflammatory conditions the amount of albumin which it contains is considerable—one to two per cent. If such an amount of albumin be found, even without the presence of specific organisms, and with the symptoms of acute leptomeningitis, the diagnosis of the latter condition is reasonably certain. But if pyogenic organisms be found the diagnosis is absolute. If there be found tubercle bacilli, the nature of the meningeal inflammation is settled beyond a doubt;

while if streptococci be found, the purulent character of the meningitis is certain. In many instances streptococci are not revealed even when the inflammation is purulent in character, but it is to be remarked that Lichtheim has recently detected their presence in two cases out of three. The number of observers who have corroborated these findings of Lichtheim is already very great, and the procedure has taken its place as a legitimate diagnostic method.

After the diagnosis of meningitis has been made it becomes a matter of considerable importance, in estimating the prognosis, to determine its variety and the location of its greatest intensity. If the character of the exudation can be made out, this will of itself be of some service in contributing to a localizing diagnosis, simple purulent leptomeningitis and meningitis in which the serous element predominates, are commonly primary of the convexities and extend to the base by a process of secondary infection. The headache, stiffness of the neck, and incessantness of the vomiting are of little service as pointing to the seat of the lesion, although it is probable that these are more distinct and intense when the inflammation is primarily of the convexities. Of equal indefiniteness are motorial irritation symptoms manifested in the extremities. On the other hand, manifestations of motorial irritation in the territory of the cranial nerves, strabismus, inequality of the pupils, grimaces, difficulty of articulation and of swallowing, the early appearance of bulbar symptoms, and intense optic neuritis indicate basal exudation of great severity.

After all has been said of the diagnosis, we may repeat that the most important factors in leading up to it are the detection of the source of the malady by an examination of those parts of the body which have been shown in the paragraphs on etiology to be the starting-points of the disease, and then the development of the symptoms, associated as they are one with the other. Although some one symptom, such as the fever, usually considered of diagnostic importance, may be absent or of slight intensity, the association of the other symptoms, and their development in a progressive manner, are the important features in the diagnosis.

Clinically it is important to consider whether leptomeningitis be primary or secondary, for this is of value as regards both diagnosis and prognosis.

Meningitis is said to be primary, although due to the pneumococcus, streptococcus, etc., when the symptoms of meningitis are not superimposed upon the clinical symptom complex of some other disease, from which it may or may not have had its origin. It is likewise said to be primary when meningitis develops in a previously healthy individual, when the primary manifestations of the causa-

tive factors are in the meninges, or in pathways directly leading to the meninges, such as the walls of a wound. In all other conditions the inflammatory process is said to be secondary; such, for instance, when the disease develops during or immediately subsequent to an attack of erysipelas, pneumonia, typhoid fever, etc., or when it is the consequence of otitis media interna, disease of the cavities and natural passages of the skull, or of infective emboli having their origin in distant parts of the body. It will at once be seen that secondary meningitis is by far the more common, and on account of the depraved state of the system which the anterior disease causes, the outcome of this form is more unfavorable. The diagnosis of the former, the primary form, is established with greater ease than is that of any secondary form except the tuberculous where the discovery of a tuberculous locus enables us to determine the character of the meningitis. The necessity of establishing this differentiation needs no further emphasis.

Acute leptomeningitis of serous or purulent exudate is readily confounded with tuberculous leptomeningitis, with abscess of the brain, with hysteria, with a complex of symptoms occurring in acute diseases in which there are some meningeal symptoms, and to which the name pseudomeningitis is sometimes given, with acute hemorrhagic encephalitis, and less frequently with meningeal hemorrhage and tumor of the brain.

As a matter of fact the diagnosis from tuberculous meningitis, if tubercle bacilli are not found in the cerebrospinal fluid obtained by means of puncture in the lumbar space, or if tubercles are not to be detected by the ophthalmoscope in the choroid, can never be positively made. The teaching that it can, for which many authors are responsible, is in the highest degree misleading. A probable differential diagnosis can be made by considering the sources of infection, the age at which the disease develops, the course of the disease, and its duration. If the symptoms of meningeal infection develop in a patient of a tender age; if the development be preceded by obscure and non-meningeal premonitory symptoms; if the onset of the disease is so insidious that it is difficult to fix upon the exact time at which they begin, and if the symptoms pursue a subacute course, with certain remissions which prompt a hope that the patient may recover, and which are in turn succeeded by exacerbations in which all the symptoms are worse than before, the disease is probably of the tuberculous form. If on examination of the body a source of tuberculous infection can be found in any of the viscera or the glands, or if the patient has previously suffered from any tuberculous manifestations, suspicion that the disease is of the latter variety should be entertained.

It should always be borne in mind that following a tuberculous infection of the meninges there is often a septic infection, and that later in the disease the secondary infective process may be superadded symptomatically to the former and so alter the clinical features of the disease. Lastly, it cannot be too emphatically stated that the presence of tubercle bacilli in the withdrawn cerebrospinal fluid settles the diagnosis beyond peradventure.

Abscess of the brain not infrequently develops from causes similar to those which are responsible for acute leptomeningitis, and when symptoms of cortical involvement are well marked in the latter disease it becomes difficult to distinguish the two, particularly as brain abscess is often associated with some meningitis. In abscess of the brain the headache in the beginning is more localized and less intense, the stiffness of the neck is neither so pronounced nor so early of development, obstinate constipation and retraction of the abdomen are not common symptoms of the disease, and the occurrence of some localized paralysis is earlier and more frequent in abscess. By many writers who use the English language, and particularly by Macewen, there is described a dissociation between the pulse rate and the degree of temperature in brain abscess, a slow, full pulse associated with an elevation of temperature to 102°–105° F., but I have never been able to verify this. Nevertheless the pulse in brain abscess rarely has the rapid, tense, incompressible characters of meningitis nor is the temperature so uniformly high. It is particularly the localizing symptoms in brain abscess which follow manifestation of purulent infection of the brain secondary to trauma or cessation of a discharge from the ear, with the early development of optic neuritis, the absence of any organisms in the cerebrospinal fluid, and the presence of an increased amount of albumin, that will lead to the diagnosis.

The simulation of leptomeningitis by hysteria is rare, especially in this country. Attention has been called to it by several European writers, lately by Bardal. In hysteria the individual symptoms of meningitis may be present, such as headache, vomiting, constipation, fever, delirium, somnolence, etc., and convulsions may occur individually or collectively, but in every instance differential symptoms of diagnostic import will be present, not to mention the history or evidences of neuropathic stigmata, inversion of the phosphates, etc., that can be elicited.

This and other forms of pseudomeningitis are more apt to be confounded with the tuberculous form, and they will therefore be considered more extensively under the latter.

It is only latterly that the symptom complex of acute non-suppu-

rative encephalitis, which not infrequently goes on to partial or complete recovery, has been recognized. Oppenheim has shown the reality of the latter and his observations are being corroborated by many observers. This disease is to be differentiated from leptomeningitis principally by the absence of pronounced meningeal irritation symptoms, such as intense and increasing headache, stiffness of the neck, etc., by the predominance of focal symptoms, one of the most prominent of which is aphasia, and by its most favorable outcome.

Occasionally the symptoms of meningeal hemorrhage come on so acutely, and the irritation symptoms, such as headache and stiffness of the neck, are so manifest that they are mistaken for acute leptomeningitis. The absence of fever (in some cases of meningeal hemorrhage the temperature is subnormal), the presence of large quantities of blood in the fluid removed by lumbar puncture, and the history of trauma or inherent defect in the vascular system of the patient, will suggest the diagnosis.

Occasionally it becomes necessary to differentiate acute leptomeningitis from all those conditions in which headache, delirium, motor irritation symptoms, and later coma are present. Such conditions as acute delirium, which occasionally ushers in acute diseases where the infection process has been overwhelming, delirium tremens, delirium gravis, uræmia, and acute ptomaine poisoning—in each of these conditions the differential diagnosis must be made by the discovery of the causes on which they are dependent, or by the isolation of pathognomonic symptoms of the last-named diseases, particularly taken in connection with the facts that none of the latter runs a gradually progressive course with the constant characteristics which we have mentioned as occurring with leptomeningitis.

Formerly it was considered necessary to give the most elaborate details of the differentiating factors between typhus fever and epidemic cerebrospinal meningitis, but the practical disappearance of the former disease from most parts of the civilized world obviates the necessity of doing so to-day.

It is between the two forms of acute meningitis, the acute purulent and the acute epidemic, that one should hasten to make a differential diagnosis. The prevalence of the latter epidemically will, of course, contribute to this end. Lumbar puncture may be utilized to make the diagnosis. If the fluid withdrawn contains the cocci of the one form or the other, and if cultures made from it produce the respective diseases in susceptible animals, the diagnosis of the variety of the meningitis will be made and the prognosis contributed to, for in epidemic cerebrospinal meningitis the prognosis is immeasurably better than in the simple purulent form.

Prognosis.

The prognosis of leptomeningitis depends very largely on the cause of the attack, and on the type of epidemic if the disease be of the latter form. In every instance the outcome must be looked upon as hazardous. The prognosis is best in the cases which occur in children and in which no specific cause except an indifferent trauma can be determined, and in the cases following on influenza uncomplicated with pneumonia. Even in these cases, particularly those first mentioned, when life has been spared there remains some sequel of serious import, such as blindness or deafness. I have seen deaf-mutism develop after an attack of meningitis which occurred before the speech faculties had become fully developed. In another case an attack of leptomeningitis occurring in childhood left the patient so that at puberty there were motorial defects which presented symptoms very similar to those of multiple sclerosis. It is probable that most of the cases of meningitis that end in recovery are either of the serous form or of the mild form of epidemic meningitis. Cases of the purulent form, and particularly those that follow on wound and focal origin of infection, that is, those in which the morbific agents are the pneumococcus and the streptococcus, are almost always fatal. When these cases go on to deep coma, and when there are associated with the coma symptoms indicating profound cerebral compression, the prognosis is of the worst.

Mistaken opinions regarding prognosis are most often the result of incorrect diagnosis. There develops with some acute diseases such as rheumatism, typhoid fever, pneumonia, scarlet fever, etc., a complex of symptoms very closely simulating leptomeningitis, and which is mistaken for it. It is the failure to recognize these as cases of false or spurious meningitis that must account for errors of prognosis.

The prognosis of the communicable form depends upon the epidemic. It is worse at the beginning than at the end of the epidemic, worse in one locality than in another, and worse one year than another. In the mild type of these cases the death rate varies from fifteen to thirty per cent., while in the severer form not infrequently the death rate becomes higher than in any other infectious disease, except some forms of tuberculosis, not infrequently reaching eighty or ninety per cent. The cases of sudden and severe onset, indicating readiness and severity of infection and the early appearance of coma, and the appearance of bulbar symptoms showing the severity of the exudation, are harbingers of early death. The duration of the epidemic form of the disease is almost as variable as the clinical type. Many cases

at the beginning of an epidemic succumb within forty-eight hours, while others, particularly of the intermittent type, extend over several weeks before convalescence is completed.

Treatment.

The most important treatment of acute leptomeningitis, whether it be epidemic or not, is the preventive. The same factors that are conducive to the occurrence of croupous pneumonia favor the occurrence of leptomeningitis, and the measures that are taken for the prevention of the one are equally inimical to the development of the other. It is particularly at times when the mortuary list of cities, or the experience of physicians in the country, shows that the occurrence of meningitis, pneumonia, and influenza is more common than usual that measures should be taken to keep up the bodily tone and especially that excessive fatigue and hygienic errors be avoided.

The next most important feature in the prevention of meningitis is the early discovery and vigorous treatment of all those conditions, particularly the local ones, to which it is secondary.

Early diagnosis is an extremely important factor in the treatment of the disease, and to this end the withdrawal of fluid from the lumbar arachnoid space is earnestly advised—not that I believe that the aspiration of fluid from the subarachnoid space can be of particular service in ameliorating the disease. If the diagnosis can be made early—and particularly a differentiating diagnosis—then the disease can be treated in a rational manner. That the lumbar puncture may some day be the avenue through which medicaments intended to reduce the potency of the inflammation or destroy its excitants can be introduced, as has been hinted by some, seems to me beyond the pale of possibility. When the diagnosis has been made, or when the presence of the disease is strongly suspected, the patient should be placed in a large, well-ventilated, dark, absolutely quiet room. The head should be shaved and it with the neck and forehead surrounded with ice-bags. If the patient is robust, and particularly if the meningitis is not secondary to some disease that has exhausted the patient, topical venesection by wet-cups at the back of the neck or general venesection is earnestly recommended. These are the most potent measures to relieve the pain next to the hypodermatic administration of morphine; and as the pain must be mitigated, these measures are preferable to the latter on account of the influence which morphine has to increase intracranial congestion.

Aside from these measures the treatment is purely symptomatic. If the temperature rises and remains above 103°, the full bath at 80°

of twenty minutes' duration, repeated every two or three hours, is to be advised, cold applications to the head being kept up meanwhile. The administration of large quantities of iodide of potassium, even up to an ounce a day, as has frequently been recommended by writers, is to be deprecated as being at variance with all that we know concerning the action of that drug and its real therapeutic uses. Small doses of the bromide of potassium administered simultaneously with a smaller dose of the iodide salt, fifteen grains of the first and from seven to ten of the latter, has sometimes seemed to me to be of service in quieting the patient and in mitigating the severity of the motorial irritation symptoms.

All forms of irritant applications to the back of the neck, the occipital region, and the mastoid processes are earnestly to be deplored. The slight amount of good which they might possibly do would be more than counteracted by the intense suffering which they, in common with all forms of stimulation, cause the patient. In the beginning endeavor should be made to get free action of the bowels, and this can be most speedily done by the administration of drop doses of croton oil, by the mouth if the intervals between the acts of vomiting permit, otherwise by the rectum.

The administration of sleep-producing agents in the beginning of the disease, after the pain has been somewhat alleviated by local or general bloodletting, or by morphine, can do no harm, and is often provocative of some sleep which not alone makes less the agony of the patient, but increases his resistance to the disease.

In the treatment of meningitis serosa Quincke has warmly recommended the administration of mercury, internally or by means of inunctions, carried up to a point of slight mercurialization, especially in acute cases. In some instances the use of salicylate of sodium in doses of from ten to twenty grains has been encouraging. If the meningitis be secondary and directly attributable to a disease, such as otitis media purulenta, erysipelas, or disease of the nose or its accessory cavities and sinuses, or if it follow injury and wound infection, treatment directed to these conditions cannot be adopted too early or with too much vigor, and always with the end in view to prevent further infection and to allow of free and thorough cleansing and drainage. In treating cases of alcoholic pseudomeningitis it should not be forgotten that the disease is a secondary exudative condition brought about directly by a pathological and asthenic condition of the circulation. Therefore measures to maintain the integrity of the circulation should be adopted early.

In cases in which it has been determined by lumbar puncture and the course of the disease that the leptomeningitis is a purulent one,

and particularly when there are symptoms of any localizing value, the question of operation must come up. It is difficult to make any general statement in reference to these cases which will be neither misconstrued nor misunderstood. It may, however, be said that in cases in which septic infection can be traced directly as from an otitis media purulenta, an infected wound, etc., the skull should be trephined as near the point of infection as is feasible, and the greatest pains should be taken to cleanse and remove the purulent products, and particularly if there be purulent foci which are causing injury by pressure. The rapidity with which purulent meningitis, starting at the convexities of the hemisphere, extends to the base and even to the cord, should be borne in mind in estimating the benefit to be derived from such an operation. It must also be borne in mind that abscess is liable to follow operation undertaken for the relief of such meningitis and likewise septic pneumonia; but a sufficient number of recoveries following operation have been recorded to make the precedure justifiable.

The general management of cases of meningitis is the same, it matters not what the pathological form may be, and it is of the greatest importance that the details of such management be observed. The pulse, temperature, and bladder should be carefully watched, and when deviations from normal which predict death appear, urgent measures should be adopted for their relief. Frequently the proper use of digitalis, of the cold bath, or of the catheter will tide the patient over some critical period until the recuperative power gains the ascendancy. The possibility of bulbar symptoms, particularly difficulty in swallowing, should be kept in mind, and proper measures taken to prevent or relieve them. When there is marked dysphagia resort should be had to nutrient enemata. More is to be gained by attention to the patient's rest and comfort and by combating symptoms as they arise than from any stereotyped plan of treatment.

Meningeal Tuberculosis.

Symptoms.

The symptoms of tuberculous meningitis group themselves under three headings, which in the order of their development are called the symptoms of the prodromal period, of the stage of irritation or excitation, and of the stage of depression and coma. Between the period of irritation and that of depression there is an interval when the symptoms are at one time aggressive, at another time regressive. To this period the name of the stage of oscillation has been given.

The prodromal period may be of long duration, weeks and even months. Its duration depends upon the severity and rapidity of infection and the resistance offered by the meninges, that is, the general resistibility of the patient. Not a few cases, on the other hand, are lacking the prodromal period, the onset of the disease having all the characteristics of an acute infection, viz., sensations of frigidity or a distinct chill, an immediate and considerable rise of temperature, and unmistakable symptoms of meningeal irritation. Usually the prodromal symptoms consist of a change in the patient's disposition and demeanor, and a gradual impairment of nutrition, both of which develop so insidiously that they are scarcely noticed by those who are in continual association with the patient until they become quite pronounced or until the advent of the second stage.

In young children the prodromal period is not so typical as in children of a more advanced age, but patients of all ages whose meninges are being infected with tubercle bacilli gradually become less forbearing to all sorts of excitation and irritation. They are intolerant of light and of noises and are loath to make any sort of sustained intellectual effort such as is required in speaking, listening, or study. Their disposition changes from lively and cheerful to morose and despondent. Diversion may be essayed in toys, in play, in books, in surroundings; they interest but for a short time and are then cast aside for something else, and this in turn for another, or for a withdrawal to some quiet, dark room, or to bury the face in the mother's lap. A feeling of continual unrest, of inability to secure contentment or diversion which is present not only during the day but at night, while sleep is disturbed and robbed of its salubrity by a persistence of the same symptoms, is the striking feature of this stage. During this period the appetite is capricious, sometimes associated with vomiting, but more often with indigestion and symptoms of gastrointestinal irritation which are frequently referred to the presence of worms by mothers and nurses, and progressive emaciation is the consequence. The tongue is generally coated, the breath is foul and heavy, and the bowels are constipated and irregular in action.

The nutrition of the patient becomes gradually impaired, and this is shown by progressive loss of weight and increasing paleness. The skin becomes translucent, and the veins which are seen beneath it with exaggerated clearness seem large and more prominent. The eyes often have a brilliant appearance, particularly in contrast with the weary, troubled countenance. Often, however, the eyeballs are expressionless, lying dull and listless in the orbits, while the color of the face is not particularly changed.

During the prodromal stage the temperature is not elevated as a

rule, but a number of cases are on record in which there has been a febrile rise of from one to four degrees. The pulse rate is variable, generally somewhat accelerated and the impulse is somewhat irregular. Occasionally I have noticed, when the patient was asleep, a marked slowing of the pulse.

Oftentimes these symptoms will all be aggravated and it will appear as though the individual is to experience an attack of acute illness; but after a brief duration the symptoms subside and the attack is then attributed to indigestion or to some indifferent cause. These remissions are very misleading.

After a time, variable in almost every individual case, the direct acute symptoms are ushered in and generally with abruptness in marked contrast to the prodromes. The parents may not have remarked the change in the child's demeanor, disposition, and nutrition during the prodromal period, but now that attention is called to them by unmistakable symptoms of profound illness, they recall occurrences which at the time had been looked upon as trifling but which now seem portentous. The early symptoms in the adult are headache, vertigo, constipation, psychical disturbances, sleeplessness, and severe vomiting. Distinct chill is of rare occurrence; in thirty-two cases carefully studied Rieder says it occurred twice, and in the first of these the beginning symptoms were right-sided paralysis and epileptiform convulsions, and in the second seventh nerve paralysis.

The symptoms of the stage of irritation are not unlike those of ordinary leptomeningitis, and as a matter of fact they cannot be differentiated at the bedside. To avoid needless repetition they will receive but scant mention here. The most striking and suggestive symptoms are four in number: headache of increasing intensity, which causes involuntary shrieks, known as the hydrocephalic cry; vomiting without nausea, simple regurgitation of the bile-stained contents of the stomach, projectile in character and of frequent repetition, and not followed by prostration; constipation of the most obstinate character, associated with such marked contraction of the abdominal parietes that the shape of the abdomen resembles the interior of a boat; and fixity and retraction of the head and stiffness of the neck.

It is to be remarked that the stiffness of the neck does not usually develop with the abruptness and severity that it does in ordinary meningitis. These symptoms develop almost simultaneously and may be accompanied, and in young children they usually are, with universal or partial clonic convulsions. As the eruption of tubercles is usually at the base, the convulsions are the expression of a general cerebral irritation and not of a specialized cortical area, although there are cases in which an isolated tuberculous agglomer-

ation in the meninges of the convexities over one or more individual brain areas, has developed in connection with extensive basal tuberculosis, causing first localized convulsion and afterwards paralysis (Boix).

I have seen two cases of tuberculous meningitis in adults, one secondary to tuberculosis of the lungs, in which the tuberculous deposit in the pia was localized to a small area at the bifurcation of the Sylvian fissure, just over the operculum, and in which the symptoms were at first convulsions of one side of the body and later paralysis of the arm and face of the side opposite to the lesion.

The spasm of the muscles of the neck is often associated with contractures or twitchings in other muscles such as the eye muscle when it gives rise to strabismus, or in the muscles innervated by the seventh nerve in which it causes grimaces or deformity. Although there is generally a flexed position of the extremities, particularly of the fingers which are clenched into the hands, there is not as a rule twitching or spasm of individual muscles or groups of muscles, or even fibrillary twitching as in ordinary meningitis. The reason for this has already been explained; it depends on the location of the tubercles. Soon after the development of the stage of irritation, changes are to be detected by the ophthalmoscope in the optic nerves. At first this has the characteristics of an intense congestion which later passes into a fully developed optic neuritis. Oftentimes the ophthalmoscope reveals the presence of tubercles in the choroid, and then the diagnosis is made certain.

Soon after the occurrence of the symptoms mentioned above, evidences of marked perversion of the senses and intellect develop. The patient, in the beginning restless, agitated, intolerant of light, noises, and all forms of disturbance, soon adopts a fixed position with the head extended or buried in the pillow, which he changes only on emission of the seemingly compulsory shriek, or when agitated by a partial or complete convulsion, both of which phenomena continue to occur even when the patient is apparently asleep. Soon the intellect becomes clouded and confused, the patient is irrational in his communications and answers, if old enough to make any. In young children manifestation of psychical irritation is referred to by the family as "flightiness," while in older patients it is evidenced by delirium, at first of not such great intensity but gradually increasing, becoming violent during exacerbations of the pain, and resulting in proportionate mental obnubilation. The appearance of the patient from the beginning of the stage of irritation is not always characteristic, although frequently there is a fixity of the features, and absence of expression. The face is pale, but often the skin is

flushed and mottled, and when the finger is drawn across the skin, not only of the face but of the body, there appears a phenomenon, first recognized by Trosseau, known as tache cérébrale. Although of common occurrence this is not pathognomonic. In some cases, particularly in those in which the symptoms are of abrupt onset and rapid development, an erythematous eruption occurs on the extremities and body, and occasionally there are patches of herpes on the lips. The eyes are firmly closed to guard against exacerbation of pain by the light, the pupils are irregularly contracted and very sluggish, if at all responsive to light, and the patient makes no evidence of recognition of substances by taste or smell.

The temperature is elevated, from 101° to 104,° of a distinctly remissive character, the time of exacerbation being governed apparently by individual conditions and associated disease. The pulse is rapid and does not differ materially from the pulse in ordinary meningitis save in tension, this being not so pronounced in this form of the disease. In a general way it keeps pace with the temperature, diminishing in frequency with the febrile remissions, increasing with the exacerbations during this period. The bedside record may show the most remarkable variations in these two phenomena almost from hour to hour; as a general rule, however, it may be said that both are most marked at night.

Soon, however, there occurs a remarkable dissociation in temperature range and pulse. This occurrence, although characteristic of the third stage or stage of depression and coma, not infrequently occurs during the latter part of the period of irritation or in the oscillatory period which follows the latter. While the temperature continues to remain elevated and to be swayed by exacerbations, the pulse rate drops ten or twenty beats below normal, while the rhythm shows a tendency to become irregular and intermittent.

The respirations are increased in frequency; but aside from this, during this stage they are not materially changed except in rare cases in which irregularity of the rhythm, a precursor of the Cheyne-Stokes type, is an early manifestation. Simon believes that a constant sign of tuberculous meningitis in the beginning is irregular rhythm of thoracic movements and inequality in the amplitude of respiratory expansion—the irregularity of respiration being shown by participation of the lower half of the thorax only in the respiratory act and disturbance of the synchronous action of the thorax and diaphragm. As yet these observations have not been corroborated.

As the stage of irritation reaches its height or end, the bodily condition of the patient begins to show the effects of the disease. Emaciation progresses gradually; the mouth becomes dry and foul,

filled with viscid mucus; the urine is diminished in quantity and has the ordinary febrile characteristics; the reflexes, which in the beginning were easily elicited and lively, now become diminished, and the contractured condition in the various parts of the body begins to relax.

When all the symptoms have perhaps reached the development that we have indicated and the outlook for the child's life seems imminently grave, a more or less complete cessation in the progress of the symptoms may ensue, or a considerable amelioration of some and a disappearance of others will tend to invalidate the correctness of the diagnosis and lead to false hopes of recovery. Remissions of such character are not so common at this stage as they are in the premonitory stage, and frequently they correspond to that brief period in which the exudate is distending the subarachnoid space and ventricles, but has not yet become so great that it gives rise to pronounced pressure symptoms. As soon as they appear the symptoms of irritation are at an end, and those of depression leading up to coma are then in the ascendancy.

It is impossible to take into account the many anomalies of development which tuberculous meningitis has. Sometimes, in children, the first symptoms may be an attack of convulsions, soon followed by hemiplegia, which may begin to ameliorate for a time, and then quite suddenly the patient passes into a condition of coma. Such a case has been reported by Money in a child nine months old. In other cases the symptoms in the beginning will be entirely focal and in their character indicative of motorial irritation. In such cases it is not until the basal exudate or the intermeningeal and ventricular pressure becomes manifest that the suggestive symptoms of the disease develop.

Eventually, after one or more of these exacerbations and remissions, so-called oscillations, the character of the symptoms assumes a type of depression, and the patient passes from the stage of irritation into one in which an ever-deepening coma is the prominent symptom. This stage corresponds probably with an increase of fluid in the extraventricular, the intraventicular, and the subarachnoid spaces. The headache disappears, the hydrocephalic cry is heard no more, the face loses its fixed, annoyed, and astonished expression, the twitchings of the muscles, the contractures of the extremities, the deviations from parallel in the axes of the eyeballs all gradually disappear, and are followed by a flaccidity of these parts, and in some cases by paralysis, particularly in the distribution of some one of the cranial nerves, the third and seventh being the ones most frequently involved. The mouth is partially open and filled with a tenacious, foul-looking mucus; the tongue is dry; vomiting has ceased; the bowels remain

constipated, but the abdomen has lost its tense, retracted appearance, it may have become tympanitic, and later in this stage it often becomes very much distended. The temperature still remains elevated, and the thermal curve has the same vacillating characteristics: higher at one hour, lower the next. The pulse becomes slow, irregular, both in rhythm and in the volume of its beats, sometimes intermittent. These two conditions form the phenomenon of dissociation mentioned above. When the patient is lying quiet, the pulse may be no more than 40 to 60 per minute, but on the slightest change in the position of the body, such as on being raised to administer medicine or nutrition, it becomes so small, rapid, and thready that it can scarcely be felt. The respiration becomes more irregular and eventually as the symptoms of bulbar involvement ensue it assumes a typical Cheyne-Stokes type. The surface of the body is now no longer mottled; there is apparently complete vasomotor relaxation. The extremities become cold and moist, and in them as well as in the face there appear evidences of defective oxygenation of the blood which point to early dissolution.

In young children—that is in those in whom the skull bones have not yet united by filling in of the fontanelles and ossification of the sutures—the enlargement of the head, particularly in its circumference, may be made out from day to day. In the adult, paralysis in one member, of the muscles supplied by one or more nerves of swallowing, or a general flaccid condition of the whole body develops, and with these symptoms ever increasing the coma continues to become more profound, and the patient expires with a typical clinical picture of cerebral asthenia. For some time before death the sphincters may be relaxed, causing incontinence of urine and fæces. The abdomen becomes distended and ballooned; the conjunctivæ are injected; one eyelid is perhaps paralyzed, the other wide open, covered at the edges with mucus which extends over the ball of the eye; the lividity of the face and extremities becomes extreme, or death may be produced by one severe convulsion, even though convulsions have not occurred for several days previously.

Etiology.

The essential cause of tuberculous leptomeningitis is the bacillus of tuberculosis. Without this the disease does not occur. The etiology then can be considered under the following heads:

I. The conditions which are favorable to, and allow the development of, tubercle bacilli in any part of the body;

II. The sources of the bacilli and the avenues by which they gain entrance to the meninges; and

III. The condition of the meninges which allows the bacilli to take up their abode therein and to excite a typical reaction.

The conditions which are favorable to and facilitate meningeal tuberculous infection do not differ very materially from those that predispose to tuberculous infection of other serous membranes, such as the pleura, peritoneum, and serous envelopes of joint cavities, except that the pia is more resistant to such infection than any of these membranes. Age is perhaps the most important indirect factor in the causation of tuberculous meningitis, as the disease occurs almost exclusively between early childhood and full adolescence, the majority of cases occurring between the first and the fifth year. In one hundred and one cases observed by Barey the youngest patient was nine months old, the average age being from three to six years. The disease is comparatively rare before the end of the first year and after puberty, although naturally cases do occur even at the tenderest age and after maturity. Its occurrence is more common in nursing children than is supposed.

The disease is slightly more frequent in the male than in the female sex, as are all other forms of tuberculosis. It is much more common in the winter and spring than during the other two seasons, on account of the facilities offered for the development and propagation of the bacilli, by the enforced housing which the temperate climate, where the disease most frequently occurs, entails, and on account of the prevalence at that time of diseases which tuberculous meningitis not infrequently follows, such as mumps, whooping-cough, measles, and more rarely scarlet fever.

The disease may occur in families in which there is no tuberculous history, and shows itself in more than one member; but more often there is a history of tuberculous disease in some of the immediate ancestors or relatives, almost all writers having remarked the frequency of the disease in children who inherit a so-called scrofulous diathesis.

All conditions that tend to lower the patient's vitality predipose to tuberculous meningitis by lessening the resistance which the general system as well as the meninges themselves offer to the infection. Study, enforced activity of the brain, particularly when coupled with depraving physical conditions, lack of exercise in the open air, defective sanitary surroundings, neglect of hygienic principles, improper and insufficient food, such as milk of an impoverished mother, or, for those in whom the disease occurs after the period of nursing, food lacking in fats, are powerful predisposing factors. Falls and blows on the head, particularly a repetition of these, and surgical operations seem in many cases to be directly provocative of an at-

tack. Many children who develop tuberculous meningitis have before their illness a decidedly precocious mental development, and the expenditure of energy in this direction seems to facilitate tuberculous infection of the meninges, particularly when the child's eagerness and tendency to study is fostered by parents and teachers. It is more frequent in the children of the foreign-born population and those in the lower walks of life than in the native born and well to do. It forms a far greater proportion of deaths in the mortuary lists of large cities than in those of small, and is more frequent relatively in urban than in suburban communities.

The disease is predisposed to by the acute infectious diseases as well by the acute catarrhal and inflammatory affections of the gastro-intestinal tract—conditions which make sudden and profound impression upon the nutrition of young children.

The sources of the bacilli in the great majority of the cases are tuberculous affection or foci in other parts of the body, such as of the lungs and pleura, of joints and bone, of the peritoneum, or of some of the glands, such as the testicle or the glands of the lymphatic system. Of these, the pulmonary organs are by far the most common sources. Weil has reported a fatal case in a girl, seventeen years old, who had never menstruated. The mucous membrane of the uterus was transformed into a tuberculous mass. The infection of the meninges is by no means always secondary to infection in other parts of the body, although in young children it is almost always so. In adults, on the other hand, primary infection of the meninges rarely occurs. The avenues by which the bacilli gain entrance to the meninges are principally the lymph current and the blood current. When the source of infection is some tuberculous focus of the cephalic end of the body, such as of the eyes, ears, nose, or throat, parts which are in connection either directly or by anastomosis with the intracranial lymphatic system, the current of lymph is the infection-carrier to the meninges. When, however, the tuberculous focus is in a distant part of the body such as the intestine or the lungs, the blood current itself is probably the infection-bearer. Tuberculous infection of the meninges, like that of the lungs and of the glands, is probably sometimes transmitted from the mother to the child before birth. Some writers (Honl) would explain most cases of primary tuberculous meningitis in this way, but it does not seem at all plausible to me. When the infection of the meninges is direct it is probably mediated through the infection of a wound or a part of the head which has direct connection with the lymphatics of the meninges. Certain it is that infection of the meninges from tubercle bacilli taken in from the outside world, without first causing

tuberculosis of some other part of the body, is of exceptional rarity, and hardly occurs except in very young children, although a case illustrative of primary tuberculosis of the meninges, occurring in a man fifty-two years old, in which the most careful search failed to reveal an old tuberculous focus, has recently been reported by Bastian. The tubercle bacillus is very slightly saprophytic and consequently has no particular development outside of the human body. The bacilli may take up their abode in one part of the body, like the lungs or the lymphatic system of the viscera, and without producing lesions attended with symptoms will be the source from which the meningeal infection arises.

Among the conditions of the meninges which make them susceptible to the tubercle bacillus, may be mentioned all the conditions that have a tendency to disorder or deprave the circulation of the brain and meninges, whether they arise within the brain as psychical, or without as the effects of trauma, alcohol, or excessive heat. The neuropathic constitution or diathesis, the heritage of which has been remarked by many writers, may also come under this heading. Blows, trauma to the head, and the like may light up a latent tuberculosis.

Morbid Anatomy and Pathology.

The appearance of the skull and dura differs according to the age of the patient when tuberculous meninigitis develops. In the very young the head is always enlarged in the way characteristic of acute hydrocephalus. In the adult the skull presents no departure from the normal. On the removal of the calvarium the veins of the diploë and the sinuses of the dura are generally very much congested. Aside from this, however, in a great number of cases there is nothing abnormal to be seen when the dura is cut through. The pia, before the brain is removed from the base of the skull, presents a shining, glistening appearance, and there may or may not be an excessive amount of fluid in the intermeningeal space. This is due to the fact that the favorite seat of the tuberculous eruption is not the pia of the convexities, although in adults the latter is not an uncommon location. When the brain is removed from the base of the skull and turned over, the characteristic granular appearance is unmistakable. These small white granules, varying from a point so small that it is barely recognizable by the naked eye to the size of the head of a pin, completely stud the meninges in certain locations, following particularly the line of the blood-vessels. Their favorite seat seems to be in the flexures of the pia which dip into the fissure of Sylvius, and in the other spaces at the base of the brain where there is an invo-

lution of the pia, such as around the optic chiasm, the anterior and posterior perforated space, and in general in the area included by the vessels forming the circle of Willis. It is not alone the granulations or tubercles which are so striking when this part of the brain is examined, but the appearance of the exudation, which may be hemorrhagic, serous, serofibrinous, or fibrinopurulent, and of a turbid, offensive yellow or yellowish-green color, which bathes these parts and is carried by the flexures of the pia into the fissures at the base and at the side of the brain.

In some cases the exudation predominates while the eruption of tubercles is very scant. In other cases the exudate contrasted with the amount of granulation is comparatively insignificant. The blood-vessels are distended and in the pia substance there are often seen slight hemorrhages, particularly if the inflammation be an acute one. The hemispheres of the brain are altered in much the same way as in acute purulent meningitis. The cortex is œdematous and swollen, so that it occupies more than its usual space, even though there is no abnormal ventricular distention. The convolutions, especially of the base, are flattened, while those of the convexities, if the ventricular effusion be not very great, present a normal appearance. Very rarely are slight hemorrhages, which are so common in purulent leptomeningitis, found in the substance of the brain. When the brain is laid open, it is very exceptional not to find the ventricles distended to some degree, and the amount of fluid which they may contain varies up to several quarts. The choroid plexus appears tortuous and overdistended, and the ependyma of the ventricles has a cloudy appearance. When the cortex is examined microscopically the most important changes found are foci of obliterative endarteritis. These evidences of arterial degeneration are apparently produced by the irritation of the tubercles in the adjacent meninges. It is altogether probable that these foci of endarteritis obliterans, and the areas of softening which form around them if they have existed long enough, slight though they are, may produce focal symptoms which, occurring early in the disease, are often misleading and difficult of interpretation. Oftentimes minute spots of softening are found in the basal ganglia; these are the result probably of the combined effects of the exudate and of obliterative conditions in the vessels.

In rare instances the examination of the brain of a patient dead of tuberculous meningitis shows very few changes aside from the mere presence of the granulations. The hemispheres appear normal, the pia is not adherent over the convexities and but slightly so over the base, and is devoid of any inflammatory appearance; the ventricles may be but slightly distended, and the fluid which they contain,

as well as that in the subarachnoid space, may be clear and not great in quantity. It is possible that in these cases a retrograde process has set in.

In other instances, the changes in the cortex, revealed by the methyl-blue and aniline-blue-black stains, are marked and easy of detection. They have recently been carefully studied by Goodall. In the cortex, just beneath the meninges, very many small round cells and numerous flask-shaped cells are seen which form a meshwork with the neighboring cells. The processes of some of these cells reach the meninges, and it is, in part at least, through them that the adherence of the pia to the brain is due. In cases in which the liquid exudation from the pia is plentiful, adhesion is prevented by the exudation. The cells of the layer of small and large pyramids are in some cases found stunted and atrophied, often only the nucleus and degenerated-looking processes being left. When the cells of the pyramids show these changes the spindle cells in the vicinity, which are probably scavenger in their functions, are increased, distinct, and prominent. The blood-vessels, particularly the minute ones, are always distended, while in many places the obliterative changes above mentioned are seen.

When the tubercles themselves are examined microscopically, it is seen that they do not differ materially from those found in other serous membranes such as the peritoneum. The miliary masses or tubercles, located in the lymph tracts and in the lymph sheaths of the blood-vessels, are the result of the action of the bacillus upon the fixed cells of the tissue, which leads to the formation of epithelial-like protoplasmic cells, which are called epithelioid cells. These little knob-like foci of epithelioid cells formed by nuclear fission or karyomitosis, are devoid of blood-vessels and are found in various stages of development or decay. The inflammatory alteration which these new formations and the bacilli cause in the adjacent blood-vessels is responsible for the character of the exudate. The vascular phenomena are not different, except in degree, from those attending inflammation of any kind. When the exudate is serofibrinous and serocellular, the number of dead leucocytes is considerable. The presence of other pathogenic bacteria such as the pus cocci may give individual characteristics to the exudate.

The variety of inflammatory products found in this disease is often explained on the ground of mixed infection, some pathologists being still unwilling to admit pyogenous properties of the tubercle bacillus. That tubercle bacilli may cause a purulent meningitis must, however, be conceded after the demonstration of Fraenkel, Lubarsch, Buchners, and others.

That a mixed infection does occur no one can deny, but it is probably not the rule. Such mixed infection may give a very different clinical aspect to the case, for the symptoms of meningeal tuberculosis are primarily basal, while those of coccal and other bacillary infection are convexity symptoms. The most common forms seen in mixed infection, demonstrable after death, are the diplococci, especially the pneumococcus. Pesina and Honl have reported a case in which the distribution and character of the pathological products were in keeping with what has been said of these two infectious agencies, the tubercle bacillus and the pneumonia diplococcus.

Course.

The course of the disease has been sufficiently indicated by the division of the symptoms according to the stages. It should here again be emphasized that this method of division is a purely artificial one, and is never seen at the bedside with the sharply differentiated features with which it is portrayed in the books. Some symptoms which are mentioned as occurring in the third stage may occur in the stage of irritation, while symptoms which are common and diagnostic of the second are often found during the prodromal stage. But this much may be said in almost every case of tuberculous meningitis: the course of the disease is insidious; the clinical symptoms are remittent and frequently misleading; and although the period of oscillation may last for a long time, the exacerbation which passes into the stage of coma is certain to occur.

Duration.

The duration of the disease varies from weeks to months, dating the beginning of the disease from the occurrence of the prodromal symptoms. It is frequently difficult to establish the beginning of meningeal tuberculosis, and particularly in cases in which tuberculosis is in a more advanced stage in another part of the body, and to which the meningeal infection is secondary. The prodromal symptoms in many cases are evidences only of the primary tuberculous involvement—whether this be of the lungs, of the intestines, of the glands, or of the bones; that is, they occur during the stage when the toxic products, the result of tuberculous aggression in other parts of the body, are producing a toxæmia and depraving the parts which the blood furnishes with nutrition until at last these become vulnerable to their destructive activity.

As a rule it may be said that the duration of the disease from the beginning of the period of irritation to death is from five to ten

weeks, frequently shorter, especially in young children, often more prolonged, especially in adults. If to this is added the period of prodromal symptoms or stage of incubation, the duration of the disease may be said to extend over several months. The period of excitation, headache, vomiting, and convulsions generally lasts from six to ten days, and is succeeded by the stage of oscillation, which is of variable length. After the stage of coma once sets in death is not delayed, except occasionally, for more than one week.

Diagnosis.

The diagnosis of meningitis is an easy one. The diagnosis of the kind of meningitis is one of the most difficult problems that can confront the physician at the bedside. We are aware that it is stated in many treatises on this subject that the diagnosis of tuberculous meningitis is easy if the patient has been under observation from the very beginning of the disease. We are willing to admit that this is true in part, and that it would be entirely true if the course of the disease were not subject to such wide individual variations, depending upon the age of the patient when infected, upon the resistance which the patient's strength opposes to the development of the disease, upon the intensity of the infection, upon the amount and severity of the tuberculosis in other parts of the body, upon the location of the tuberculous eruption in the meninges and the extent of this eruption, upon conditions which it causes in the vascular supply of the brain, and upon a large number of other conditions. It is infinitely more difficult to diagnose tuberculous meningitis from other forms of meningitis, from that caused by the diplococcus pneumoniæ for example, than it is to differentiate the different forms of pneumonia. We have, however, had during the past decade added to our diagnostic aids a measure which in large part robs the disease of this difficulty—a measure which can be applied with facility, which is particularly devoid of danger, and which may have a slight tendency to influence the course of the disease. We refer to the procedure of Quincke known as puncture of the subarachnoid space and which we have described in detail in another part of this article. Although the presence of tubercle bacilli has not been demonstrated in several cases of meningitis which the autopsy showed were of a tuberculous character, it has been found in upwards of fifty per cent. of the cases that have been reported. Hereafter when the technic of searching after the bacilli becomes more widely known and practised the percentage of cases in which they may be found will probably be materially increased.

This not only furnishes a means for diagnosing tuberculous meningitis but it is of the greatest service in diagnosing the tuberculous cerebrospinal meningitis from ordinary cerebrospinal meningitis, a diagnosis not easily made at the bedside. Examination for the tubercle bacilli taken from fluids which have accumulated in other parts of the body may likewise be of service—for instance, examination of a pleuritic exudate. A case illustrative of this has recently been reported by Fraenkel. The patient, a man, became comatose after walking to the hospital. The presence of a right-sided pleuritic exudate was revealed on physical examination and corroborated by puncture. An examination of this fluid under the microscope showed it to be simply an emulsion of fat drops. No cocci were found. Such findings are considered characteristic of tuberculosis, even though the bacilli are absent, for the latter may be present in spore form or in too small numbers. On autopsy an extensive tuberculous meningitis and tuberculosis of the lungs were found.

Other means for the positive diagnosis of tuberculosis have already been mentioned, such as the discovery of tubercles in the arachnoid. Second only in importance to this is the detection of tuberculosis in other parts of the body. If this be made out, and if the symptoms of meningeal tuberculosis be superadded to them, the diagnosis of the latter disease is practically a certainty. But without the presence of some one of these, the diagnosis must remain at least somewhat problematical, and particularly is this true in cases in which tuberculosis of the meninges is localized and produces the symptom complex indicative of focal disease, and only later on the symptoms of the general condition.

A condition of acute exhaustion of the nerve centres which sometimes follows on gastrointestinal disorders of children, is that to which the name spurious hydrocephalus was given by Gooch, Marshall Hall, Abercrombie, and others, who recognized the condition at a time when meningitis was considered almost synonymous with hydrocephalus. The term is now disappearing from medical literature and these cases are more often called pseudomeningitis. As we have already said, the symptom complex of pseudomeningitis develops sometimes with acute fevers such as rheumatism, typhoid fever, etc. In such cases it simulates acute leptomeningitis, serous or purulent. When it develops after acute and chronic disease, particularly gastrointestinal, it is often difficult to differentiate it from tuberculous meningitis. The symptoms of this form of pseudomeningitis are sometimes spoken of as dividing themselves into stages. The first is that in which the infant is restless, irritable, feverish, and sleepless, or the sleep is of a restless, moaning character. The appetite is lost, the

bowels are flatulent and loose, and the patient becomes emaciated from day to day. The child is excessively irritable, cries violently, and gives the impression of great suffering. After these symptoms have persisted for a somewhat indefinite time the patient passes into a sort of stupor and coma. At first it can be aroused from the state of drowsiness, but when aroused it remains indifferent to surroundings and quickly relapses, and later it is impossible to awaken it. The pulse becomes slow and irregular, the respirations are sluggish, the stools are offensive and voided in bed, the eyes are dulled with pupils dilated, the temperature which at the beginning of the condition was elevated now becomes subnormal, and the patient passes from the state of collapse and coma to death, if the symptoms remain unresponsive to treatment.

Very often it is with the greatest difficulty that this condition is differentiated from tuberculous meningitis of nursing children. If a child sickens with the above symptoms after some acute disease which has become chronic and which is of an exhausting nature, such as gastroenteritis; if the symptoms develop progressively and if exhaustion and prostration go hand-in-hand with the emaciation and diarrhœa; if symptoms of involvement of structures at the base of the brain do not enter into the clinical picture; if there be no evidence of tuberculosis in other parts of the body, not to mention many other symptoms which will indicate tuberculous infection, the diagnosis of pseudomeningitis will be readily arrived at. If meningeal symptoms similar to these develop, as they sometimes do, with rachitis, the diagnosis must be made only after the pathognomonic accompaniments of the latter, particularly in the bones, are demonstrated. Even then it should not be forgotten that tuberculous meningitis may occur in children who have rachitis.

The presence of worms in the intestines of young children often causes cerebral symptoms not unlike those of some stage of tuberculous meningitis, especially the irritative stage. These are irritability, peevishness, disturbed and restless sleep, vomiting, constipation, slow, irregular pulse, and convulsions. The transitoriness of the symptoms, the readiness with which they yield to remedies directed especially to the intestinal tract, the presence of worms in the stools, the absence of increasing headache and muscular rigidity, particularly of the neck, and the apyrexia—all speak against the dependence of the symptoms upon tuberculosis of the pia mater.

A number of other conditions may be confounded with tuberculous meningitis of children, such as the meningeal symptoms of dentition, of influenza, of rheumatism, etc., but attention to the three cardinal symptoms of this form of meningitis, which are present when the

disease is developed, as well as many which are nearly pathognomonic when occurring in connection with others, makes the diagnosis not a difficult one.

In the adult the symptomatology of tuberculous meningitis can scarcely be paralleled by that of any other disease if the former be at all typical. The nearest approaches to it are septic meningitis and hysteria. The former has already been considered. A careful examination which reveals the stigmata and dissociated symptoms of the latter will be sufficient.

When the tuberculous infiltration of the pia is predominantly over the convexities, or when the symptoms are dependent upon focal tubercles and not miliary tubercles, then the symptoms can be those of almost any focal disease and are not to be positively differentiated from the latter, although some attempt may be made by an inquiry into the individual tendency to tuberculous disease. Further than this it is impossible to go unless tubercle bacilli can be found in some fluid or tissue of the body.

Prognosis.

Time and experience have established the belief that the tuberculous form of meningitis is almost always a fatal disease, but there are a number of cases on record in which it would seem that the diagnosis was correct, in which recovery has followed either spontaneously or as the result of some therapeutic procedure. Such cases have been reported by Dujardin-Beaumetz, who saw improvement in a case of meningitis in which tubercle of the choroid was demonstrated by the ophthalmoscope, so-called cerebroscopy of Bouchut. Freyhan has reported a case in which the presence of tubercle bacilli was revealed by examination of the cerebrospinal fluid obtained by lumbar puncture, in which the patient made a slow recovery. A most convincing case has recently been reported by Janssen. The patient had a typical attack of tuberculous meningitis and recovered under the administration of large doses of potassium iodide (nine hundred grains a day). Three years later the patient died from pulmonary tuberculosis and the reparative lesions of the ancient infection in the meninges were found. Many cases of recovery, both spontaneous and after operation, have been reported, in which the diagnosis had been made from the clinical signs but had not been substantiated by other evidence. Such cases of spontaneous cure are mentioned by Bristowe, Henoch, Hellier, Osler, Jacobi, Ord, Gorse, and others. Under treatment we shall have again to refer to some cases in which the same result apparently has been obtained by operation. It is probable that a very small percentage of the cases of tuberculous

meningitis in children terminates in latency, and in a small proportion of this number the latency may be so complete that the word recovery may be properly applied to it. But it must also be said that cases of undeniable infection of the meninges by the tubercle bacillus in adults terminate always in death.

Treatment.

The treatment of tuberculous meningitis is the most unpleasant phase of the disease to consider. Results of treatment are epitomized by what we have said under prognosis. As in every other disease of which the termination is almost invariably in death, innumerable plans of treatment, medical and surgical, have been adopted; but today, after they have all had a fair trial, the physician stands in the presence of this disease with the same feeling of inability to shape its progress as did his predecessor of a generation ago. Nevertheless the fact that cases have been recorded of recovery, in which there was no reason to doubt the correctness of the diagnosis, and which have been attributed to certain plans of treatment, makes it fitting that in every case an active plan of treatment should be begun early and pushed vigorously.

The general directions given for the care of ordinary meningitis apply as well to the tuberculous form. The essential point of difference in the treatment of these two diseases is that in the latter it is recommended to give large doses of iodide of potassium, from fifteen to forty grains three or four times within twenty-four hours, and during the stage of irritation to combine with this small doses of bromide. It has been recommended that mercury be rubbed into the neck and behind the ears, but this as well as all forms of counter-irritation has never been shown to have the slightest virtue. Contemporaneously with the appearance of almost every one of the coal-tar products, substances which not only relieve pain but reduce temperature, has been the report of their service in the treatment of tuberculous meningitis; but aside from their symptomatic use they are of no value. It is known that iodoform in solution when injected into the peritoneal cavity has caused a cure of peritoneal tuberculosis, and this has led to the considerable use of iodoform in tuberculous meningitis. Lemoine gave eight grains of iodoform dissolved in ether for a period of three months, without toxic symptoms, and reports favorable results. Warfvinge reported five cases successfully treated by inunction of iodoform to the scalp. As the result of his recommendation several physicians of Stockholm employed it, but all with the same result—failure; so we are forced to the conclusion that the majority if not all

of his cases in which favorable results of treatment were reported, were not genuine examples of meningeal tuberculosis.

The operative procedures that have been suggested for the cure of tuberculous meningitis are also numerous, but up to the present time their employment is scarcely more than justifiable, so slight has been their influence on mortality. The object of operation in tuberculous meningitis may be either palliative or curative, the first to reduce pressure, the second to facilitate the conditions which will bring about fibrous change in the tubercles and inactivity of the bacilli. The operations that have been recommended are simple trepanning and tapping the ventricles, or tapping the ventricles through a fontanelle if it be open, trepanning and drainage of the ventricles, and draining the ventricles and subarachnoid space through an opening in the vertebral column. The first plan has been abundantly tried by American surgeons, Keen, Agnew, and others, as well as by Bergmann, Morton, Franks, Pollosson, abroad, but has never been successful. Lannelongue trephined in four points of the skull, two in front and two behind, and then passed through on the pia mater a liquid or gaseous current; the patient succumbed rapidly. Recovery has been recorded following opening of the subarachnoid space in the cervical region by Ord and Waterhouse, but the fluid was not examined for bacilli, and there was no positive evidence of tuberculosis. A more convincing case is one of Fürbringer's reported by Freyhan, in which after lumbar puncture and withdrawal of fluid in which tubercle bacilli were found the patient slowly recovered. Quincke has seen cases which have been decidedly ameliorated by the same procedure. It is difficult to conceive how this operation can be of any particular therapeutic utility unless future experimentation can show that the puncture furnishes an opening through which substances are carried into the intermeningeal space which exercise a wholesome influence on the tuberculous eruption and the exudate, which seems scarcely probable. The question of operative interference in tuberculous meningitis may be summarized in a few words. The only procedure that is justifiable at the present day is lumbar puncture, and that cannot be advised with any promise of cure, but it is often of benefit in ameliorating the severity of the pressure symptoms.

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SARCOMATOSIS OF THE PIA.

Sarcomatosis of the pia may occur in a diffuse or circumscribed form apart from similar new growths in the central nervous system. It is so infrequent that it constitutes a pathological rarity. Cases, however, have been described by Coupland and Pasteur, by Busch, by Cramer, and others. Mention only will be made of the condition here for the sake of completeness, as it has no pathognomonic symptoms. The disease consists of angiosarcomatous new formation in the shape of innumerable, circumscribed, little white patches which develop in the pia of the brain and spinal cord, and are distributed variously and irregularly over the superficies of the central nervous system. In some instances they have been found in the substance of the nervous system, but they are not infrequently confined to the pia. In such a case the new growth does not extend into the brain substance, although it may subject the latter to some pressure.

The disease seems to occur most frequently during the years of childhood, more rarely in early adult age. The clinical symptoms which the disease produces are those of meningitis: headache, vomiting, stupidity, manifestations of increased intracranial pressure, and the rest.

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CHRONIC MENINGITIS.

Chronic inflammation of the pia occurring as a separate disease is not a common condition. When it does occur the disease process is rarely, probably never, limited to the pia alone, but involves the superficies of the cortex as well, thus constituting a meningoencephalitis. The starting-point of the inflammation is, however, in the meninges and not in the cortex. In one very well-defined nervous dis-

ease (general paresis), this process in a diffuse form constitutes the cerebral lesions of the disease. In general paresis, in addition to the chronic diffuse meningoencephalitis, there are, however, unmistakable changes throughout the entire nervous system, so that the process in the cortex and cerebral meninges constitutes only a part of the morbid anatomy of the disease. It is therefore not considered here, as this chapter is confined to a consideration of chronic disease of the pia which gives rise to clinical entities. In a general way chronic meningitis may be classified into true and false chronic leptomeningitis. Etiologically considered the first may be subdivided into syphilitic (by far the most frequent), tuberculous, senile, and simple. Chronic pseudomeningitis is always caused by the chronic, intemperate use of alcohol. In the strict sense of the term it is not a meningitis at all, but a condition of œdema and simple exudation and infiltration, although naturally there may be associated with it and dependent upon the same cause well-marked changes in the blood-vessels. The œdema and opacity of the pia in this condition of the meninges and brain are most marked at the convexity along the longitudinal fissure. Similar changes are usually found in the dura. The changes in the cortex are not secondary, as was at one time thought, to those of the pia, but are the expression of the malign effects of alcohol on the cortical blood-vessels, and through them on the tissue.

Chronic leptomeningitis is a very rare condition, but of its varieties the most common by far is that due to syphilis. It constitutes one of the forms of brain syphilis, and would very properly be treated under that caption. According to its location syphilitic meningitis may be divided into meningitis of the base and meningitis of the convexities, the former being by far the more common. Both of these develop occasionally in a somewhat acute fashion, but their ordinary course is chronic or markedly subacute. The acute form is so very rare that it will merely be mentioned.

Syphilitic Meningitis.

Location and Pathological Product.

The seat of the syphilitic process is principally at the base and in the middle fossa of the skull. This fossa receives the middle cerebral lobe and other important brain structures, and gives points of support, conduction, and exit to the optic chiasm and optic nerves, the third, fourth, the ophthalmic division of the fifth, the sixth nerve, and some filaments of the sympathetic nerves, as well as important vessels like the middle meningeal artery. In basal syphilitic menin-

gitis the interpeduncular space especially is the seat of syphilitic exudation and new formation. Many of the most prominent symptoms of basal syphilitic meningitis are due to involvement of the structures above enumerated, so that it is important to bear them in mind. The pathological product of syphilitic meningitis does not differ from that of other syphilitic lesion of soft tissues except in so far as it is modified by the structural elements of the meninges. The essential element of the lesion is the granulation tissue, which consists of round cells closely pressed together, supported by the connective-tissue structure of the pia. When this granulation tissue is collected into definite masses, it matters not how small or how large, varying in size from a pin's head to a closed hand, it forms a gumma, and the presence of such masses embedded in the pia constitutes gummatous leptomeningitis. The process is usually a diffuse one and the conglomerations of granulation tissue form little nodes or masses. In some instances these little nodes embedded in the meshes of the pia have a resemblance to a tuberculous eruption. Collections of them are often found around nerves like the optic and oculomotor and intimately attached to their sheaths. When the round-cell infiltration is disseminated into the substance of the meninges, filling up its meshes and alveoli, and the granulation tissue is not collected into masses, it constitutes exudative syphilitic meningitis. Not infrequently both processes are to be seen immediately adjacent to one another, the diffuse infiltration in the area around the isolated gumma. The characteristic feature of the syphilitic process in every case is the development of round cells. Very rarely are other cells, spindle and giant cells, found in the seat of inflammation in sufficient number to be conspicuous; and when they are they can probably be attributed to other causes, such as a superadded tuberculous infection being responsible for the giant cells.

The newly formed granulation tissue is studded with unstable blood-vessels, which with the granulation tissue give the very recent gummatous formation a grayish-red appearance. Later on, however, on account of contraction of the vessels and the accumulation of cells about them, their calibre becomes obliterated and they are choked out of existence; this gives the mature lesion a characteristic vascularly impoverished appearance. This immature vascularization is originally called into existence by the impetus given to blood-vessels of the tissues, and is the result of budding proliferation of these vessels. The connective tissue of the structures into which the exudation takes place also manifests a reaction to the irritant in the shape of increased production of the fixed cells. These changes in

the vessels and in the connective tissue are terminal ones, for they eventually contribute to their own destruction by assisting in the process of necrobiosis which goes on *pari passu* with the development of granulation.

The retrograde metamorphosis which always takes place in this unstable new formation as well as in the supporting tissue is that characteristic of fatty degeneration, transformation into mucous tissue and simple atrophy, and does not require special description here. The introgressive changes in their various stages give rise to the different naked-eye appearances which syphilitic meningitis presents. In some cases the meningeal area which is the seat of luetic disease will be soft to the touch and of a grayish-white color or slightly tinged with red, or if there is a gumma the color may vary in different zones of a cross-section. In other cases the tissue will be of a dirty grayish-yellow appearance and caseous, almost liquid to the touch, with serous infiltration and congestion of the blood-vessels around it, particularly in the adjacent depressions or sulci. If the retrogressive changes have been of slow occurrence the consistence of the newly formed tissue is well preserved. The naked-eye appearance of syphilitic leptomeningitis is particularly striking in the basal circumscribed or diffuse variety. Frequently the brain can be removed from the base of the skull only with great difficulty on account of adhesions of the pia to the brain and the basal structures, to the dura, and possibly to the skull itself.

After it is removed the interpeduncular space, the region of the chiasm, in short any part of the structures in the middle fossa may be embedded in and destroyed by the thickened, adherent, degenerated pia. The adhesions to the dura, and through it to the bone, may be so intimate that they cannot be separated. To the brain the pia is adherent over large areas, or here and there, and united to it by proliferation and overgrowth of the new tissue into the brain substance. Naturally these conditions of pia cause destructive changes in the substances of the brain which, however, vary in each case.

The vascular changes in the pia and the brain, aside from those in the newly formed tissue in syphilitic meningitis, are often very well marked. They require, however, no separate description, as the changes are the well-known ones of luetic arteriocapillary fibrosis. It is not at all improbable that the occurrence of some of the symptoms which stand out so prominently in syphilitic meningitis, particularly in the basal form, such as vertigo, are immediately dependent upon such vascular disease.

Symptoms.

The symptoms of syphilitic meningitis are irregular in development, inconstant in manifestation, and vary according to the location and extent of the syphilitic process. Syphilitic leptomeningitis of the base is attended by symptoms which often do not form a part of the clinical picture produced by circumscribed luetic disease of the pia over the convexities. The symptoms of basal meningitis will be mentioned first. It is impossible in this article to point out in detail the difference between circumscribed and diffuse basal meningitis. The extent and confines of the lesion will have to be inferred from the number and association of the symptoms.

The most invariable symptom is headache. Its location and characteristics are not absolutely constant, but yet are looked upon as somewhat pathognomonic. The pain is a dull, boring ache, which often increases to almost unendurable agony. It is frequently continuous, worse at night than in the day, and aggravated by stimulants, excitement, excesses, and by mental application. It has no characteristic localization, being sometimes at the base of the skull and back of the head, sometimes across the head from ear to ear, and very often not localized at all, the entire head being seemingly the seat of the pain. Dizziness is a very distressing symptom, not only during the exacerbation of the pain, but in the intervals. At the height of the pain or during an exacerbation of the vertiginous symptoms there is often vomiting of great severity. It is not preceded or attended by nausea, and is not relieved by evacuation of the stomach; its characteristics are those of cerebral vomiting. Symptoms of irritability, intolerance of trifling annoyances, incapacity for sustained mental effort, vacillation of purpose and of thought early betray the development of the disease. With or without the occurrence of these mental symptoms there may be more or less prolonged periods of drowsiness, hebetude, and even of unconsciousness varying in intensity from a state from which the patient may be aroused to one of complete coma. These attacks of mental sluggishness and coma sometimes come on very early in the disease; they may be of considerable duration, and may clear up quickly even when the patient is not under treatment.

After the less alarming of these psychical symptoms have existed for some time, or after the occurrence of one or two severe attacks, it will often be remarked that the patient's perception-readiness is not so acute, that memory is more treacherous, that interest and attention are difficult of arousal, that in short a slight degree of dementia

is present. Indications of changes in the blood-vessels are often not lacking, and it is probable that many of these symptoms as well as the motor-irritation symptoms to be mentioned presently are genetically associated with this vascular degeneration. Such symptoms are cold, clammy extremities, depressed animal vitality, impaired digestion, functional sluggishness of the bowels, and the secretion of a large quantity of pale urine of low specific gravity. This excess of urine may be so great and constant that it constitutes a polyuria, which is very apt to be associated with polydipsia. Heightened arterial tension and diminished calibre and elasticity of the blood-vessels can often be made out by examination of the pulse.

Motor-irritation symptoms such as muscular twitchings, localized or general convulsions, and transient or permanent mono- and hemiplegia, sometimes constitute a prominent feature of the symptom complex of basal syphilitic meningitis. Motor symptoms due to affection of the third and seventh pairs are frequently among the early symptoms. They may consist of twitchings in the orbicular muscles of the eyes or the frontalis, very rarely twitchings of the emotional muscles of the face, and more rarely still of the masticatory muscles, or of some paretic manifestation like lid-droop. The pupils are often irregular, and sluggish reaction on directing a ray of bright light on the retina is one of the most constant accompaniments. A transitory squint may be the first manifestation of cranial-nerve involvement. These spasms and paralyses are not conjugate and associated, like those from cortical lesion, but affect individual nerves or parts of nerves, and in the beginning are especially characterized by vacillancy.

Some disturbance of vision is usually not long delayed if the luetic process at the base is not extremely limited and remote from the optic tracts, chiasm, and nerves. These visual shortcomings, consisting of obscuration of sight, hemianopsia, and even complete amaurosis, depend very largely on the part and extent of the visual tracts involved, and thus upon the extent and location of the lesion. Their intensity may often be inferred with considerable accuracy from ophthalmoscopic examination. The ophthalmoscopic findings may be choked disc, optic neuritis, descending neuritis, or primary optic atrophy, and their relative frequency probably corresponds to this order of enumeration. Choked disc, which is always bilateral, suggests an extensive basilar lesion; while optic neuritis, which is often unilateral, bespeaks involvement of the sheath of the intracranial optic nerve. When disturbance of vision, such as hemianopsia, hemichromatopsia, or loss of vision in some segment of the visual field, or

amaurosis of one eye and hemianopsia of the other, exists without in the beginning detectable changes in the background of the eye, then the syphilitic process is causing destructive action behind the chiasm in the optic tracts or possibly in the chiasm itself, and neuritis and descending atrophy are to be constantly sought after, for although their occurrence may be long delayed they rarely fail to make their appearance eventually. Affection of the optic nerves is often bilateral, although it may appear first in one eye then in the other. Very remarkable is the fact that, like all the other symptoms of basilar syphilitic meningitis, it may entirely clear up to recur again, or possibly when the process has not gone beyond choked disc with the aid of treatment it may disappear forever. Such a desirable termination of choked disc has been noted a number of times, and I have recently recorded an instance.

The cranial-nerve affections which next to the optic are numerically the most frequent are those of the third nerve. The point of exit of this nerve is anatomically so situated that a basilar meningitis of comparatively slight extent is very apt to involve not only the nerve of one side but of both, and all the branches of each, as well. The importance of recognizing the frequent syphilitic origin of paresis of the oculomotor nerve has been pointed out by many writers, but none has done it so forcibly and so thoroughly as Uhthoff. In the large number of cases which this writer has analyzed and personally observed, by far the most common lesion which produced disease of the oculomotor was basilar syphilitic meningitis, and in this opinion he has received fullest confirmation from Oppenheim, Marina, Siemerling, and many other recent writers. The symptoms produced by involvement of the third nerve by the syphilitic process is that of single or double ophthalmoplegia, as all the branches of the oculomotor nerve are usually involved. In the beginning the paralysis may show itself in but an individual structure supplied by the third nerve, such as the eyelid when it is manifest by ptosis; but advance in the course of the disease is generally attended by a progressive involvement of other branches of this nerve, although the paralysis may remain confined to a single muscle. External ophthalmoplegia is a rare accompaniment of syphilitic basal meningitis. There can be no doubt, however, of its occurrence. Formerly it was thought that this variety of ophthalmoplegia spoke unequivocally in favor of involvement of the nucleus of the nerve, but paralysis of single branches of the oculomotor nerve with basal meningitis has been observed by so many different observers that the reality of its occurrence is beyond doubt. Symptoms denoting injury of other of the anterior cranial nerves occur, but they are by no

means so common as those just mentioned. Of these the trigeminus is more frequently involved than the patheticus or abducens.

The symptoms which accompany involvement of the fifth nerve are limited to those occurring through injury to its sensory branches, anæsthesia, hyperæsthesia, analgesia, formication, etc., in the ocular and cutaneous supply of the first and second divisions. If the meningitic process involves the olfactory nerve the symptoms will be disturbance of its function, particularly manifest by anosmia. If the sheath or trunk of the facial nerve be implicated there will be convulsive or paralytic phenomena in the muscle supply of this nerve, that is facial tic or facial paralysis. The cranial nerves given off posterior to the seventh are by virtue of their anatomical position involved with relative infrequency. An exception should possibly be made of the pneumogastric nerve, as symptoms of injured cardiac innervation such as tachycardia, bradycardia, and inconstancy of rhythm and volume of the pulse are frequently a part of the clinical picture of basilar syphilitic meningitis. It will be seen that the symptoms of cranial-nerve involvement are among the most important in the symptomatology of basilar syphilitic meningitis. They are not more constant, nor so much so, as headache, vertigo, and slight disorder of the mental faculties. They are, however, more significant, for careful study of their development, their course, and their combination have led clinicians to attach the greatest diagnostic importance to these symptoms. The association of symptoms is itself very suggestive, but more so than this is the variation in presence and intensity of the symptoms. This striking intermittency in the severity of the symptoms has been spoken of by almost every writer since Murri called attention to it in 1876. Sachs especially has emphasized the diagnostic importance of this change in the intensity and course of the symptoms.

Symptoms of motor irritation such as localized or general spasms or convulsions of the extremities are not essential integers in the clinical manifestations of basilar syphilitic meningitis, nor is monoplegia or hemiplegia, although any of these symptoms may occur and the first named occurs with some frequency. The convulsions are not focal and they conform to no special type. They are resultant in all probability upon disease of the blood-vessels and not upon the basilar meningeal process directly. These convulsive attacks of an epileptoid nature may be accompanied by loss of consciousness, or they may alternate with periods of restricted consciousness which approximate in character the so-called dreamy state.

A paralytic condition that sometimes develops with basilar syphilitic meningitis is alternating hemiplegia, paralysis of the face

on one side and of the extremities on the other. This syndrome, when developing with other symptoms of basilar meningitis, especially the headache and the optic and oculomotor nerve involvement, indicates secondary invasion of the peduncle by the luetic process. In Uhthoff's monograph twelve such cases are collected from the literature. Very rarely do other lower segments of the central nervous system, such as the pons and oblongata, become involved by extension and proliferation of the syphilitic process into them, but when they do pontal and bulbar symptoms will be added to those already detailed as characteristic of syphilitic basilar meningitis.

Symptoms of Syphilitic Leptomeningitis of the Convexities.

Circumscribed syphilitic meninigitis of the convexities is much more uncommon than the form just considered, syphilitic basilar meningitis. It cannot be said that any particular area of the cortex is the seat of the lesion, although the meninges over the frontal and parietal lobes are the favorite locations. Histologically and anatomically the lesion does not differ from the basal variety. In area it may be circumscribed or diffused and in intensity it may be accompanied by a considerable degree of encephalitis or by pachymeningitis. The little granulations, so-called miliary gummata, are perhaps less common in this locality than at the base, and thickening of the pia by the interstitial exudate is more common.

It is unwise to attempt to separate these two forms of meningitis by any hard-and-fast lines, for not infrequently the process at the base extends to the convexities and thus constitutes one disease entity. In fact, in those cases of basal syphilitic meningitis in which motor-cortex irritation symptoms are prominent it is very probable that such extension has taken place. In discussing the form of meningitis at the base it was stated that round-cell infiltration and vascular change which is so striking in the meninges often extended into the cortex and thus a meningoencephalitis resulted. It is in luetic meningitis of the convexities and particularly in the circumscribed variety that this is common. In fact, syphilitic disease of the pia without changes in the cortex is the exception. When the relation of the pial blood-vessels to the cortex is borne in mind and it is remembered that in syphilitic leptomeningitis the blood-vessels are always diseased, the association of encephalitis with syphilitic meningitis will be readily understood.

The symptoms of syphilitic meningitis of the convexities differ from those of the basilar variety only by the absence of some striking symptoms of the former, particularly those of involvement of the an-

terior cranial nerves, and by the prominence of others, such as those indicative of lesion of definite parts of the motor cortex.

The headache, vertigo, nausea, irritability, mental obscuration, stupidity, etc., with the characteristics already mentioned, occur with the same frequency as in the basilar form, and may be the only symptoms for a long time. The headache especially may be a symptom which antedates by a long time the onset of any of the others, although not necessarily so. Stieglitz has reported an instance in which the patient did not suffer from headache until partial paralysis had developed. The pain is frequently confined to a very small area of the skull, and often such an area is tender to pressure and percussion. Sooner or later if the lesion be over the central convolutions motor-irritation phenomena in the shape of convulsions, epileptoid in nature, or paralysis with characteristics called cortical will occur. The predominant feature of the convulsive attack is that it is focal, Jacksonian in type. The spasm which may or may not be preceded by a definite aura begins in a separate member or digit and may be confined to it for a number of attacks and not attended with loss of consciousness; but eventually the convulsive phenomena, although definite and local in their first manifestations, become universal, and the attending loss of consciousness may be very prolonged. These attacks of focal epilepsy are of varying frequency, but like all the other symptoms their course is progressive. The paralytic manifestations—monoplegias and hemiplegias—which occur when localized areas that are posited as definite motor centres are invaded, need not be described in detail. It must be mentioned, however, that one characteristic of cortical motor paralysis, the tendency to disappear or to greatly improve, is lacking, while a state of contracture is very apt to attend or follow. Heightened myotatic irritability, manifested by increased ankle, knee, and triceps jerk, and diminished cutaneous reflex are usual accompaniments. Sensory manifestations do not cut a very prominent figure in the symptomatology of the disease. Paræsthesia, or pain of central origin manifesting itself in individual parts or members of the body, may be complained of, particularly in association with the convulsive attacks. Loss of dexterity and perversions of tactual and position sensibility have been noted when the pathological product is confined to the motor areas. These are symptoms mediated through involvement and disease of the cortex, and are not, strictly speaking, meningeal symptoms at all.

Symptoms indicative of cranial-nerve involvement do not form a part of the clinical picture caused by circumscribed syphilitic leptomeningitis of the convexities, although sluggishness of the pupils is oftentimes to be made out. When they do occur, it signifies involve-

ment of other parts—the meninges of the base, the brain tissue, or the nerve sheaths themselves.

Naturally, if certain areas of the brain to which are allocated definite memories, such as the visual, auditory, and speech areas, are injured, symptoms which bespeak such involvement, as some form of aphasia or disorder in the interpretation of impressions by such centres, will occur. Such symptoms are not of common occurrence. In this description the fact that syphilitic meningitis and meningoencephalitis of the convexities is most commonly found over the frontal and frontocentral region of the brain has been kept in mind, and the symptomatology corresponding to lesion of such parts has been delineated. Occasionally the severity of the syphilitic process is expended on the meninges and cortex of the frontal or occipital regions alone, and in such cases symptoms indicative of disorder of function of these parts may be prominent. Space does not permit enumeration or discussion of these symptoms, but they will be readily understood by reference to the section on localization in the article on Diseases of the Brain. The mental symptoms, however, which sometimes occur with luetic leptomeningitis of the convexities deserve special mention. They may be entirely wanting, but unfortunately in a considerable number of cases there is a progressive diminution of mental faculties simulating and sometimes mistaken for general paresis, while on the other hand defective inhibition of impulses and disordered association may be so great as to constitute genuine mania.

Diagnosis.

The diagnosis of syphilitic meningitis of the base or of the convexities is ordinarily made without difficulty. One of the most weighty factors to suggest the diagnosis is a history of syphilitic infection, but still more suggestive in contributing to the diagnosis is the course of the symptoms. The symptom complex itself of headache, boring, agonizing, and nocturnal; vertigo; mental shortcomings; excitability and volubility; attacks of lethargy and somnolence; and in the basal variety symptoms of oculomotor, optic, and other anterior cranial-nerve involvement, and for the convexity variety symptoms of motor and prefrontal irritation, is nearly pathognomonic. The multiplicity and diversity of the symptoms speak louder than anything else, except the irregular and episodic evolution of the disease, in favor of the diagnosis. The therapeutic test is unfortunately of service in but a few diseases, but this is one of them, especially if it is applied early. There can scarcely be any chance for error of diagnosis in a case with paralysis of one or more branches of the oculomotor nerve, headache,

vomiting, stupidity, psychical limitations, etc., which responds with comparative readiness to a course of inunctions or to a number of mercurial injections.

Course.

The course of syphilitic meningitis, if uninfluenced by treatment, is, like that of all other forms of brain syphilis, essentially a progressive one. It must not be understood from this that the disease is uniformly progressive; on the contrary, its course is characterized by periods of acute intensification, by prolonged maintenance of the *status quo*, by remissions in the severity of all of the symptoms, and by total disappearance of some. Notwithstanding all these the disease, when untreated, and occasionally when treated in the most approved way, tends month after month and year after year to become more deeply and firmly established and further removed from recovery. In meningitis of the convexities, as the disease continues to develop and the symptoms become more general in their manifestations, the clinical picture may deviate in its finer delineation from that which we have attempted to portray. Sometimes the symptom complex points only to dementia, to mania, or to epilepsy. In the basilar form, particularly if the structures at the anterior portion of the middle cerebral fossa alone are involved, there may be apparent cessation of the symptoms. This is more likely to occur after such development of the disease is reached that the structures implicated, as some of the anterior cranial nerves, are practically destroyed. But eventually after years, perhaps, there is implication of some part of the central nervous system, particularly through syphilitic disease of the blood-vessels, and pathological conditions develop which quickly lead to death.

Duration.

The duration of syphilitic meningitis is extremely variable. It depends somewhat on the time that has elapsed since the primary lesion, on the severity and extent of the disease process, as well as on the symptoms pointing to implication of the cortex and individual structures by the exudation and new growth, and to some extent on the treatment. It is not a self-limiting disease. Without treatment its course is progressive up to the point of destruction of the tissues and structures in juxtaposition to it, but not necessarily up to the point of death.

Prognosis.

The prognosis is better for meningeal syphilis than for any other form of lues cerebri. It is better in meningitis of the convexities than in the basilar form. It is better when the symptoms indicate involvement of the anterior part of the middle brain fossa than when they point to involvement of the posterior portion. In the latter case implication of the oblongata or the nerves to which it gives superficial origin is an omen of evil import. The occurrence of symptoms that point to extension of the syphilitic process to the adjacent brain tissue, as to the peduncles in the basilar form and to the cortex in the convexity form, materially lessens the chances either of recovery or of great amelioration.

Gravity of prognosis is increased when the symptoms point to syphilitic disease of the blood-vessels. A patient who has syphilitic meningitis, be it of the base or of the convexities, and who has at the same time syphilitic endarteritis, may experience some amelioration of the meningeal symptoms, but he cannot recover.

The prognosis is most favorable in those cases which occur within five years after the initial affection and in which mercurial treatment has not been thoroughly carried out. It is particularly favorable in such cases if the symptoms be entirely meningeal. It is not an uncommon experience to have a patient with fully developed symptoms of basilar meningitis, including profound affection of the oculomotor nerve and a considerable degree of papillitis, regain full and normal health under proper antisyphilitic treatment. These cases are probably all of the kind in which the predominant product of the syphilitic process is exudative and in which the round-cell infiltration does not go on to organization and later to retrogradation. The prognosis of syphilitic meningitis is worse in those persons who are addicted to any form of vicious excess, it is worse in those past middle life than in young adults, and in a general way it may be said that it is unfavorable in proportion to its duration. No such relation exists between the intensity of the symptoms and the prognosis. In some cases the symptoms, purely meningeal, may be of great severity and yet the disease terminates in complete recovery. When the symptoms and objective signs point to severe cortical involvement, the prognosis for complete recovery is unfavorable, but for marked and continued amelioration of the symptoms it may still be very good. When cortical tissue is once destroyed, however, there is no repair, although there may be partial restoration of function through the compensatory activity of other parts. The prognosis in

syphilitic meningoencephalitis varies according to the location and amount of the destructive process.

All things considered, the prognosis in syphilitic meningitis is more favorable than in any other form save acute non-infectious meningitis.

Treatment.

The treatment of syphilitic meningitis does not differ from that of syphilitic disease of other parts of the body, and consists in maintaining bodily nutrition and in the administration of mercury and iodide of potassium. The method of administering these two substances varies somewhat according to the length of time between the initial lesion and the onset of meningeal symptoms, and according to the amount and kind of treatment that the patient has had since the time of infection. In a general way it may be said that the further removed the meningitis is from the time of specific infection the less amenable will the symptoms be to mercury alone.

One of the most striking manifestations of the efficacy of mercury in syphilitic affection is seen in the rapidity with which symptoms of meningitis, especially irritation symptoms such as headache, disappear under the use of mercury. The mode of administering mercury is less important than determining the dose for the individual and then maintaining it. Personally I prefer inunction of the common unguentum cinereum applied in the customary way. Very frequently I have seen abrupt cessation of meningeal symptoms after a few applications of blue ointment behind the ears and at the base of the skull. I am at a loss for rational interpretation of the apparently increased efficacy of mercury in many early cases of brain syphilis when thus applied, but experience compels me to recognize it as a fact. Iodide of potassium should be administered, not, I believe, in combination with mercury, so-called "mixed" treatment, but separately, in a mildly alkaline water such as Vichy, and in large doses. Although it may be given contemporaneously with the mercury, if the symptoms are not of great urgency I prefer to follow the mercury with the iodide. It scarcely need be mentioned that all forms of alcoholic and narcotic indulgence are to be studiously eschewed. I have found, however, that when it is necessary to administer the potassium iodide in large doses and for a prolonged time symptoms of iodism may be most satisfactorily combated by giving from six to eight ounces of very dry sherry in the twenty-four hours. Any considerable physical effort is to be advised against, and mental application as well as all kinds of tasks and experiences that upset the patient's equanimity are to be avoided.

Treatment at home may often be supplemented with great advantage by a visit to any of the many American and European springs and baths which have gained repute for their salubrious effects on syphilitic affections. In meningitis of the convexities, after the more acute symptoms have subsided under the influence of mercurial and iodic treatment, the question of operation for removal of the scar-like formation of connective tissue which remains and which often produces focal symptoms will arise. English surgeons (Horsley, Macewen, and others) advise such procedure, but the experience of American neurologists and surgeons is not entirely in favor of it. When such a lesion is entirely unamenable to medicinal treatment and the course of the focal symptoms is progressive, everything is to be gained, I believe, by resort to operation.

Cerebrospinal Syphilis.

In the same way that the term brain syphilis (see page 111) almost always means syphilitic leptomeningitis, and is used synonymously with it, the term cerebrospinal syphilis usually implies the existence of a syphilitic leptomeningitis of the brain and the cord. When syphilis manifests its peccant activity on the central nervous system and its coverings, the disease manifestations are not usually limited to individual parts of it; on the contrary, they are likely to be distributed over wide areas. Thus luetic disease of the cerebrospinal type is much more frequent than syphilis of the brain or cord alone. This on first thought may not seem entirely true, and particularly if the teachings of the older writers are remembered. As a matter of fact, however, many of the cases formerly considered as forms of brain syphilis are shown not infrequently on the post-mortem table to be examples of cerebrospinal syphilis in which the spinal symptoms were so masked and overshadowed by the cerebral symptoms that they went unrecognized. This is readily understood when we remember that in the great majority of cases of cerebrospinal syphilis the symptoms of brain involvement are first and most marked, and although they may remit, they are always present throughout the entire course of the disease in some degree, while the spinal symptoms are almost always secondary in point of time.

Symptoms.

Syphilitic disease of the cord and its meninges is, compared with cerebrospinal syphilis, relatively rare. When it does occur it gives rise to the clinical types of transverse myelitis, syphilitic spinal paralysis, and sometimes the Brown-Séquard symptom complex. With

these alone we are not here concerned, and we shall consider them only as they enter into the clinical picture of cerebrospinal syphilis.

The mode of development of cerebrospinal syphilis is exceedingly variable, but as a rule the cerebral symptoms appear first and continue to predominate. They are usually those attending luetic leptomeningitis, such as headache, vertigo, undefinable dread, feeling of impending disaster, psychical disturbance varying from slight stupidity and forgetfulness to hallucinatory delirium and considerable dementia, convulsive phenomena, disturbance of articulation, disturbance of vision and perversion of function of some of the ocular muscles, particularly the third nerve, apoplectic attacks, and transitory, recurring, or persistent paralysis. These symptoms may develop very gradually or in part only, and then subside or become more or less stationary, or they may come on with considerable abruptness, reach an alarming intensity, and then rather suddenly remit, or symptoms pointing to involvement of other parts of the central nervous system may be added. The spinal-cord symptoms are those which may be referred to irritation of the posterior and anterior spinal roots and to implication of the medullary tissue itself by pressure of the luetic meningeal exudate, and its extension into the substance of the cord. If the intensity of the meningeal exudate and new growth be expended on the meninges of the posterior surface, sensory irritation phenomena or symptoms will be in the ascendancy. These consist of stiffness of the neck, if the meningitis be of the cervical region, a very common location; pain, radiating in the course of the intercostal nerves and lancinating in character; thoracic and abdominal girdle sensation, rhachialgia, fixedness of the shoulders and arms, subjective hyperæsthesia, and sensitiveness to pressure of the upper portion of the body. If the meningeal exudate be predominantly of the anterior surface (a very rare condition), motor irritation phenomena, spastic and spasticoparetic symptoms, and atrophy will be forthcoming. The symptoms expressive of involvement of the substance of the cord, by compression of the exudation, extension of the new growth into the substance of the cord, and coexisting destruction, vary likewise with the part of the cord involved, but will take the form of a transverse myelitis, Erb's type of syphilitic spinal paralysis, crossed hemiplegia and heminæsthesia, or muscular atrophy, according to the seat and intensity of the syphilitic process.

It is particularly the association of some of these spinal-cord symptoms with symptoms pointing to involvement of the brain that is so suggestive of cerebrospinal syphilis. So true is this that some clinicians teach that the multiplicity of symptoms, postulating as they

do lesion of parts which have widely different anatomical and functional relationships, is the most characteristic feature of the disease. Another very striking feature in the development of the disease is the decided tendency which the symptoms show to temporary remission or retrogression, which is always followed by a recurrence of the symptoms in a severer form or by the development of new symptoms pointing to involvement of other structures.

The very great number of symptoms, or the peculiar combination and dissociation of symptoms, that arise with cerebrospinal syphilis cannot be here considered individually. The cranial nerves most frequently involved are the second, third, fourth, fifth, sixth, and seventh, the symptoms of whose involvement have already been enumerated in the chapter on luetic leptomeningitis. Recurring paresis of the oculomotor nerve and pupillary inequality, particularly loss of reflex pupillary mobility, and paradoxical pupillary contraction, such as contraction in the dark and dilatation in the light, are to be considered very significant of syphilis. Occasionally ocular palsies precede the development of other symptoms for quite a long time—perhaps a year. The occurrence of these lesions with mental and physical symptoms, such as dementia, apoplexy, or hemiplegia, indicating disease of the cortex of the brain and the motor projection pathways, or with any of the symptoms of spinal-cord involvement which we have enumerated, should at once lead to the suspicion of cerebrospinal syphilis.

The association of hemiplegia with paraplegia, so-called triplegia, is a combination very suggestive of syphilis cerebrospinalis. Headache, convulsive attacks, cranial-nerve palsy, and hemiplegia associated with such symptoms as incontinence of urine, functional disturbance of the rectal sphincter, slight disturbance of sensation of the lower extremities, loss of sexual potency, and a spasticoparetic condition with increased myotatic irritability of the lower extremities, are incontestable evidence of cerebrospinal syphilis.

Not infrequently the beginning symptoms of cerebrospinal syphilis are referable exclusively to the spinal cord and are of a tabetic or transverse myelitic type. In the beginning and during the early course of the disease it is impossible to differentiate these forms from genuine tabes and transverse myelitis, but the advent of cranial symptoms is an indication of the true nature of the disease. Cases which had symptoms of ordinary tabes, so-called cervical or high tabes and pseudotabes, which continued throughout life without appearance of cerebral involvement, have been published by Brasch, Eisenlohr, Sachs, Oppenheim, Minor, and many other writers.

It has been said that oftentimes the symptoms of brain disease

entirely overshadow those of the spinal cord. This is shown most interestingly by a case recently published by Cassirer:

A fifty-year-old woman who had for some time complained of headache was seized with right-sided hemiplegia, which developed the usual accompaniments of spastic paralysis: articulatory speech disturbance; inequality of the pupils and stiffness of the right pupil; slight left-sided ptosis; inconsiderable right-sided facial paralysis; excitability, and dementia, all of which continued without material change until death. The only feature worthy of remark on examination was that the tendon jerks on the unparalyzed side were exaggerated. The autopsy revealed the presence of pachymeningitis over the pons and cerebellum and extension leptomeningitis over the crura cerebri, pons, oblongata, cerebellum, and cervical and dorsal cord. The exudation was that typical of syphilis: small, round-cell granulation masses which showed caseous degeneration in the centre and fibrous degeneration at the periphery. In the pons and left cerebral peduncle this new formation had extended in and almost completely destroyed the entire left pyramidal tract, and the left intramedullary root of the patheticus, while on the right side a small part of the pyramidal projection was destroyed. In the oblongata corresponding changes of secondary descending degeneration were found in the pyramidal tracts and in addition there was degeneration of the right spinal trigeminus. The points of entrance of the posterior roots and the posterior root zones were degenerated in the cervical cord, and the spinal, basilar, and bulbar arteries showed the most profound degeneration—mesarteritis and periarteritis.

Diagnosis.

The early diagnosis of syphilitic disease of the central nervous system is urgent because of its readier response to treatment than lesions of different constitution which present analogous symptoms.

The questions answer to which should influence one most in making a diagnosis of cerebrospinal syphilis, or any other form of syphilis of the nervous system, are: 1. Has the patient had an initial lesion? 2. Are there any unmistakable manifestations of syphilis to be discovered, such as the serpiginous syphilide (*la signature de la syphilis* of Ricord)? 3. Are there or have there been certain bone affections, such as localized periosteitis, perforation of the nasal septum, of the palate, etc., and which have developed apart from trauma? 4. Are there cutaneous and mucous-membrane discolorations and cicatrices which we know are commonly caused by syphilis? 5. Is there adenoid hyperplasia, particularly of the post-cervical and epitrochlear glands? 6. Is there history of repeated miscarriage without attributable cause in the patient or in the patient's family?

Serious investigation of each of these points and the collated re-

sults taken in connection with, first, the mode of development of the disease; second, the association and interrelationship of symptoms; third, the course of the disease; and fourth, possibly its response to antisyphilitic treatment, will enable us to make the diagnosis in more than nine-tenths of all the cases.

It is not necessary to lay stress upon the fact that many patients who present unmistakable evidences of syphilis deny all knowledge of primary infection. Such denials are often honestly made by patients who have contracted the disease in a manner out of the ordinary and who perhaps had the primary ulcer in an unusual place, while on the other hand they are not infrequently made, especially by women, with intent to deceive and so to avoid suspicion of wrong-doing.

Prognosis.

The course of the disease has been sufficiently indicated in the description of the symptoms. Progressiveness is its striking characteristic, but the progression is not uniform. At one time the symptoms threaten dissolution; at another time they seemingly point to complete recovery, a termination which does not often occur, although such amelioration of the disease may follow that the patient is restored to a life of comparative usefulness.

In short, it may be said that the prognosis in cerebrospinal syphilis is immeasurably better than that of any other disease which is attended with symptoms approaching these in severity or extent.

Treatment.

The treatment of cerebrospinal syphilis does not differ from that of syphilitic meningitis. The most important treatment is the prophylactic. Thorough treatment of the primary infection and interdiction of customs and habits which we know to be active in facilitating the development of syphilis of the nervous system are of great importance. When symptoms of spinal-cord involvement are conspicuous, in addition to the treatment of cerebral syphilis, the most assiduous care should be given to the maintenance of the function of the bladder and bowels, and the general hygiene of the patient.

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PACHYMENINGITIS.

Pachymeningitis is the term applied to inflammation and inflammatory degeneration of the dura.

It is classified, according to the layer of the dura which is the seat of the inflammation, into external and internal pachymeningitis.

Pachymeningitis Externa.—Inflammation of the external layer of the dura is a disease which the neurologist rarely sees. Its occurrence is uncommon, almost always in connection with disease of the skull bones, such as periosteitis and osteomyelitis the result of trauma and infection which bring it within the province of the surgeon. It is only when pachymeningitis extends and involves other structures that it produces irritation or compression symptoms which may be considered important neurologically. In a few instances it is probable that a chronic inflammatory condition of the external layer of the dura may be caused by trauma, which is insufficient to produce surgical disease of the bone. This affection of the dura may, however, lead to secondary change in the calvarium of an osteosclerotic or an osteoporotic nature. The former is rare, and the latter is so extremely uncommon that but a few instances have been recorded, notably by Lannelongue and by Thompson. If the process in the

dura remains limited to the external layer, it produces no suggestive train of symptoms, and a diagnosis, if made at all, must be made by exclusion. If a patient who gives a history of trauma to the skull afterwards develops a persistent headache of varying but of considerable severity, which is associated with tenderness on deep-seated pressure and on percussion, and all other causes of chronic headache can be excluded, the disease may be suspected, and perhaps the suspicion may be so well founded that one is justified in recommending the operation of trepannation. Traumatic disease of the external dura is, however, rarely confined to this layer alone. Either the inflammatory process extends to the internal layer or there develops on the inner surface of the external layer a dense, false membrane, studded with angioblasts and immature blood-vessels which sooner or later rupture. The consequent accumulation of blood and the coincident involvement of the internal layers constitute a form of pachymeningitis hæmorrhagica interna.

The fact that pachymeningitis externa tends to extend and thus cause disease, it may be purulent, of the internal layer, and in some cases of the pia as well, necessitates that active measures be instituted to overcome it before it produces brain irritation and pressure symptoms. In many instances such measures must be applied when the diagnosis is largely conjectural.

The necessity of removing all causes that are productive of the disease or that assist in keeping it up is apparent. Almost always these factors are remotely anterior and have ceased to be active. If such evidence of previous injury as indentation of the cranium exists, trepannation should be resorted to. Ordinarily, when such a condition is not discoverable and the symptoms are of a chronic nature, the treatment will consist of counter-irritation by means of the actual cautery applied to the back of the neck or over the seat of localized pain, and frequently repeated; while at the same time such resorbents and antiphlogistics as iodide of potassium and the salts of mercury are given internally. Mental rest and physical ease are important.

Pachymeningitis Interna.—This form of pachymeningitis, as an uncomplicated condition, is comparatively rare. Pathologically, it has been classified as occurring in four forms: 1. Pseudomembranous; 2. Purulent; 3. Hemorrhagic; 4. Tuberculous.

The *pseudomembranous* variety occurs as a complication or extension of external pachymeningitis and is, like it, due to trauma and disease of the cranial bones. The formation of the false membrane is the result of inflammatory reaction to the irritant. It has the vascular characteristics of all false membranes, and it may be directly

contributory to hæmatoma of the dura, or hemorrhagic pachymeningitis.

The *purulent form* never occurs as a distinct entity, but as a part of the diffuse and extensive purulent process in the other layer of the dura, its sinuses, and in the pia, which takes place sometimes with severe septic infection. It does not give rise to symptoms different from those of septic leptomeningitis, or if it does, they are so obscured by the latter that they are unrecognizable.

The *tuberculous form* never occurs as an isolated affection. When it occurs in association with other forms of intracranial tuberculosis it offers no symptoms which cause its presence to be suspected.

Hemorrhagic pachymeningitis, hæmatoma of the dura mater, or arachnoid cyst, is the most common form of internal pachymeningitis, and relatively is not an infrequent condition in certain chronic degenerative diseases of the brain, such as progressive paralysis of the insane, in hereditary or Huntington's chorea, in senile dementia, and accompanying a pathological state of the brain which is empirically termed atrophy of the brain. Sutherland has called attention to its occurrence in scorbutic children.

In the present evolutionary stage of our knowledge of this disease it excites a pathological rather than clinical interest, for as yet it is impossible to more than suspect its existence during life. Regarding the mechanism of its occurrence, two very distinct and opposite views are held. As far back as 1856 Virchow taught that the essential antecedent of the collection of blood was an inflammatory state of the inner layer of the dura, which allowed the formation of a thin membrane studded with blood-vessels, to the rupture of which the blood clot was secondary. The formation of the clot in successive, stratified layers he explained by the occurrence of repeated and successive hemorrhages. The false membrane, which separates the collection of blood from the arachno-pia, is a fibrous deposit from the hemorrhages.

The views of Virchow have been seconded and taught by many of the most trustworthy pathologists, notably by Ziegler, by Delafield and Prudden, by Fitz, and by Osler, and are accepted by the majority of neurologists, who concede that a large proportion of cases of non-traumatic pachymeningitis interna are purely inflammatory in origin. They believe, however, that subdural hemorrhage may result from other conditions.

Virchow's teachings have been combated at home and abroad, particularly by alienists and pathologists of insane asylums, who contend that pachymeningitis hæmorrhagica is not pachymeningitis at all, that the disease is a misnomer, and that the real pathological condi-

tion antecedent to the hemorrhage is of the pia, and consists principally of changes in vasomotor tone of the pial blood-vessels, followed by structural changes which allow of diapedesis of the blood.

The most ardent advocates of this view are Sperling and Huguenin in Germany, Wigglesworth and Clouston in Great Britain, and Dercum, Hoyt, and others in this country. Much evidence has been brought forward to substantiate their view, but it succeeds only in proving that a subdural collection of blood constituting a hæmatoma may occur without coexisting inflammation of the dura—a fact which all writers admit. One theory which has been recently proposed, but which has very little to commend it, is that the immediate antecedent of the hemorrhage is a vasomotor disturbance, the result of spasm and contraction of the brain vessels from a sudden lowering of intracranial pressure.

The morbid anatomy of hæmatoma of the dura requires only brief mention. It should be kept in mind that the dura is made up of two very closely woven layers of connective tissue, in which ramify the blood-vessels, the arteries on the outside, the veins in the middle, the capillaries forming a very rich network just beneath the endothelial surface of the dura. On the inner surface of the dura there is a thin subendothelial layer of connective tissue. The extravasation, occurring usually from the capillary vessels, spreads itself over the inner surface of the dura, and becomes encapsulated or surrounded by a limiting membrane. After a variable time the extravasation coagulates and causes a deposition of fibrin in well-ordered layers, which a second or a third instalment of the hemorrhage may cause to be laid on in a characteristic lamellated way. Such a hæmatoma may contain from two to twelve ounces of blood in various stages of transformation, and cause by its size and amount profound irritation and compression symptoms.

Hæmatoma of the dura is found most commonly over the vertical and parietal portions of the brain in the vicinity of the falx cerebri, although other parts, such as the frontal and basal regions, are sometimes the location of collections of blood. Robertson has clearly shown that they are of two kinds. In one they are the result of a single hemorrhage in the subdural space; in the other they are associated with extensive degeneration of the dura. When the condition is the result of hemorrhage the entire hæmatoma does not form at one time save in exceptional instances. This gives the solid part of the mass not only its arrangement in layers, but causes it to show different stages of histological transformation. In the slighter grades and manifestations of the disease, the anatomical features are limited to the formation of a tough, vascular membrane on the inter-

nal dural surface, which, on being stripped off, reveals innumerable spots of hemorrhage and hemorrhagic extravasation. There may be a number of such layers in different stages of organization, the one in juxtaposition with the dura ancient and decolorized, the innermost one recent and red. Sometimes from this internal and recent layer a profuse hemorrhage between the dura and pia, called intermeningeal apoplexy, occurs. In the variety in which degeneration of the dura predominates, the pathological change in the endothelial cells of the dura is of a proliferative and destructive nature. And this degeneration occurs not alone on the cells of the surface, but also in the endothelial lining of the perivascular canals. The nuclei of the cells undergo fatty degeneration and vacuolation, which gives this surface a peculiar glimmering appearance. The walls of the blood-vessels are thickened and in some instances the seat of distinct fatty degeneration. Occasionally there are, in areas where the blood-vessels are much degenerated, budding and proliferation of capillary vessels, and these areas may be made out to be the seat of slight hemorrhages.

Etiology.

The most important etiological factors, so far as known, have been mentioned above. In addition to the cases that occur in the degenerative brain diseases in males of advanced age, the disease also occurs in infancy, probably due to continued or violent compression of the skull during birth, from trauma, and, very rarely, in adult males who have been addicted to great and continued alcoholic excess, which causes degenerative disease of the heart, the kidneys, and the blood-vessels. Other causes leading to vascular depravity may contribute to its causation by producing vascular degeneration, such as syphilis and the acute infectious diseases; rarely does it occur with the hemorrhagic diathesis and its genesis.

Symptoms.

We shall not discuss under this heading cases of pachymeningitis hæmorrhagica and their sequelæ occurring in infancy, nor shall we refer to the traumatic cases in adults which constitute a purely surgical condition, but shall limit our description to the apparently spontaneous, idiopathic form of the disease.

As in every other intracranial disease, the symptoms depend largely on the seat of the lesion, on the rapidity of its development, and on its extent. The most uncompromising symptom is headache. The headache has no pathognomonic features. It may be very in-

tense, of a depressing, aggravating nature, or it may be just annoying, of long existence, and alternating with stupidity. Other common symptoms of meningeal irritation are not prominent. With or without having previously complained of headache, the stricken individual may manifest symptoms of motor unrest and mental irritability and excitation, which may go on to general uncontrollable twitchings, to epileptiform convulsions, limited to one member or one side of the body, which may or may not be followed by rigidity and, later, paralysis in the same area, while the mental symptoms resemble those of acute mania or acute delirium.

In some instances the condition of somnolency and stupidity increases up to complete unconsciousnes, which is then accompanied by stertorous breathing, slowness of the pulse, contraction of the pupils, cold, clammy extremities, and other symptoms of intracranial compression. Monoplegia and hemiplegia may be entirely absent, and it is rather characteristic that the cranial nerves always remain free. The prodromal and early symptoms differ from those attending other forms of intracranial hemorrhages by the more gradual onset of the symptoms and by the protractedness of the stage of irritation. After the occurrence of such symptoms as we have mentioned, the intensity of the conditions may abate, but they are certain to recur. Even when the patient has been in a state of coma, the remissions may be so complete that the patient may respond to questions and the various forms of irritation a short time afterward. The bodily temperature is usually elevated, and this has been suggested as an important differental diagnostic factor between it and intracerebral hemorrhage by Bourneville and Charcot. It is not a reliable one.

Following on the beginning symptoms of an attack there often develops a symptom—rigidity of an extremity—to which is attached considerable diagnostic weight. It is an expression of irritation of the motor centre of that extremity, which may later be manifest by convulsion or paralysis.

When an attack is ushered in with profound symptoms, such as unconsciousness, the hemorrhagic process is profuse, and the patient may pass into a stage of gradually deepening coma, with its customarily associated symptoms, to death.

Some writers, such as Fürstner, have mentioned the occurrence of unilateral nystagmus and optic neuritis, but if they do occur sometimes, they are not of the slightest diagnostic worth, and Huguenin has reported a case of extravasation into the sheath of one of the optic nerves without the development of choked disc.

When the attack develops in a patient suffering from general paralysis or delirium tremens, as it does so frequently, the symp-

toms may be so obscured by those of the latter disease that they are scarcely noticed, or if so, not recognized until repeated convulsions, rigidity, or paralysis, combined with rise of temperature and pressure symptoms, suggest the probability of its occurrence and the advent of dissolution.

Course, Duration, and Prognosis.

The course of the disease is essentially a chronic one, characterized by remissions and exacerbations, and the forms that we have been considering lead invariably to death, although that issue may be long delayed, months and even years. It sometimes becomes necessary to differentiate the symptom complex from that of leptomeningitis and from intracerebral hemorrhage. The absence of stiffness of the neck and involvement of the cranial nerves, taken in connection with the fact that the disease develops under entirely different conditions from any form of meningitis, will make the diagnosis. We have already enumerated the symptoms that will assist in differentiating it from intracerebral hemorrhage.

Treatment.

Therapeutic resources are powerless to stay the course of the disease. Something may be done, in alcoholic subjects and in those suffering from degenerative diseases of the circulatory system, to prevent its occurrence.

When recognizable symptoms occur, the general plan of treatment is the same as in ordinary apoplexy. Absolute quiet, brisk catharsis, ice to the head, vascular sedatives, and counter-irritation to the extremities may be used, but with little hope of anything more than temporary alleviation.

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MENINGEAL HEMORRHAGE.

Meningeal hemorrhage, apart from purely traumatic forms, is of exceedingly uncommon occurrence. We have in another place considered two forms, meningeal hemorrhage of the new-born or hemorrhage into the arachno-pia, and hemorrhagic pachymeningitis, also known as hæmatoma of the dura. This leaves for separate consideration here only hemorrhage into the meshes of the visceral pia and between the pia and the cortex, a variety to which the term intra-arachnoid and subarachnoid, or meningeal apoplexy, is usually given.

Subarachnoid Hemorrhage (Meningeal Apoplexy).

Etiology.

Every factor that contributes to arterial degeneration is a predisposing cause to this form of hemorrhage. The principal ones are gout, rheumatism, syphilis, alcohol, chronic indigestion, and the infectious diseases. It occurs almost exclusively after maturity. Occasionally it occurs in young children who are the victims of profound and increased malnutrition, the cause of which cannot be discovered.

The exciting causes of meningeal apoplexy are the same as those of cerebral apoplexy—mental and physical strain and trauma; in short, any condition that causes increase of heart action and blood pressure.

Symptoms.

Degeneration of the meningeal blood-vessels dependent upon constitutional disorder does not occur apart from vascular degeneration in other parts of the body, and particularly from disease of other parts of the brain. This fact explains why the prodromal symptoms of meningeal apoplexy are so dissimilar in different cases. In one they may be those suggestive of cortical irritation from the aneurysmal dilatation, an intermediary condition which takes place in cerebral blood-vessels before they become diseased enough to rupture.

Generally speaking, the premonitory symptoms are those of arterial sclerosis or arteriocapillary fibrosis; the most common being vertigo, mental sluggishness and forgetfulness, impaired digestion and lack of physical vigor, depression and an undefined feeling of impending disaster, drowsiness and sluggishness when sitting or moving about, often associated with sleeplessness, alertness, and fleeting cerebration at night, and paræsthesiæ of the head or extremities in the area of some of the cranial nerves; one or all of these may be antecedent to the symptoms produced by rupture of the meningeal blood-vessels. They are not especially indicative of threatening meningeal apoplexy they may point to the danger of occurrence of cerebral apoplexy. In former years they were often considered an entity, and labelled cerebral hyperæmia or anæmia. Very rarely do premonitory motor-irritation symptoms either of the cortex or of some motor cranial nerve occur. Yet in a few cases symptoms of motor unrest, which is akin to convulsions, and even convulsions themselves, have been noticed.

The actual symptoms attending rupture of a meningeal blood-vessel depend on the amount of extravasation and its location. Generally, however, the symptoms are those of a stroke, followed by loss of consciousness, during which unilateral convulsions may occur, but hemiplegia almost never. The duration of unconsciousness may be very brief, or the "stroke" may occur without being accompanied at first by unconsciousness. Oftentimes the unconsciousness occurs in a series of attacks, each one of which is supposed to correspond with a fresh accession of hemorrhage. These are the cases in which death often takes place shortly after complete unconsciousness is reached. Often, in cases in which consciousness is lost, the patient soon recovers enough to answer questions, to inquire what has befallen him, and to move. This corresponds to the time between the rupture of the blood-vessel which causes the shock symptoms, and the extravasation of blood which causes the irritation and pressure symptoms, and which soon follow the symptoms of returning consciousness if the extravasation be at all extensive. Frequently the patient passes, after the second lapse of consciousness, through stages of ever-deepening coma, in which an extremity or one-half of the body may be seen to be slightly convulsed, very rarely paralyzed, to death. In other cases in which the extravasation of blood is not so extensive and is confined principally to the meshes of the visceral pia and the interpial space, the symptoms will be those of increasing stupidity, mental sluggishness, and irritation symptoms, such as motor unrest, and possibly convulsions.

If the patient preserves consciousness he will complain of head-

ache, blurred vision, and indistinct hearing which will b attended by subnormal temperature for the first day or two following the shock, and then by a slight febrile rise to 100°–101° F., and often a premortal rise of considerable degree. Some disturbance of respiration, in rhythm or in volume, is almost always present. The pulse, in addition to the characteristics given to it by arterial degeneration, is usually slow.

The patient's countenance has in the beginning a striking pallor, but if the hemorrhage is sufficiently copious and extensive to cause unconsciousness, the facial circulation becomes sluggish. One of the most striking symptoms next to the repeated accessions of gradually deepening unconsciousness is deviation of the axes of the eyes. The deviation is conjugate, the eyes turn towards the side of the lesion. Next in diagnostic importance to this, and a symptom which has frequently been noticed, is deviation of the head towards the side in which the hemorrhage is located. Both of these symptoms are an expression of irritation of the motor areas for movement of the head and eyes. Persistent and enforced position of some part of the body other than the eyes and head has sometimes been noted. If the hemorrhage occurs over other specialized areas of the cortex, which it rarely does, there will be corresponding symptoms.

Morbid Anatomy.

This form of meningeal hemorrhage is the result in almost every case of the rupture of an artery; only very rarely does the hemorrhage come from a vein or a sinus. The blood, therefore, of a bright-red, arterial color is found in a semifluid or coagulated state, depending upon the remoteness from the time of its occurrence, in the meshes of the pia, in the subarachnoid space, over the convolutions, and in sulci. If the hemorrhage was of sudden onset and of large quantity, so that coagulation could not take place as fast as the blood escaped from the vessel, the great mass of it will have gravitated to the base of the brain, and will have distended the subarachnoid space which at the base constitutes the oblongata-cerebellar cistern.

In cases in which the ruptured vessel is smaller and the hemorrhage is not so great, some blood may be found in the basilar subarachnoid space; but the greater quantity will be over the convexities, where it causes a flattening and pressed-out appearance of the latter.

Blood-vessels whose coats rupture are always in a moderately advanced state of degeneration. The immediate antecedent condition is that of aneurysmal dilatation, the remotely antecedent one a fatty

and atheromatous degeneration. These changes in the blood-vessels can be readily made out on microscopical examination.

In a few cases, on cutting into the brain, it is found that the ventricles are the seat of a collection of blood, and opening the spinal canal also occasionally shows the presence of blood in the subarachnoid space.

Course.

The course of the disease, as has been hinted, is most often an intermittently progressive one. Occasionally the attack comes on gradually, the unconsciousness is a symptom of gradual development, and death soon follows its full development.

Duration.

The duration is very variable. It stands in intimate connection with the intensity of vascular degeneration and the amount of vascular extravasation. In some cases death follows very rapidly the occurrence of the hemorrhage. In other cases, if the hemorrhage be slight and disease of the blood-vessels not far advanced, the extravasated blood may undergo coagulation, retrograde metamorphosis, resorption of the liquid elements, and lymphoid encapsulation of the remains. In such cases the duration of the affection may be protracted, and at the same time there are few symptoms pointing to the presence of the ancient rupture.

Prognosis.

The prognosis of meningeal apoplexy is unfavorable, particularly for the reason that all non-traumatic intracranial hemorrhages are uniformly of bad prognosis—because they are dependent upon blood-vessels so diseased that they cannot long maintain the nutrition of the brain. Some cases may recover for a time, but death, and that not long delayed, is the usual termination.

Diagnosis and Differential Diagnosis.

The most important symptoms contributing to a diagnosis of meningeal apoplexy are symptoms of slight shock, followed by mental and physical enfeeblement and gradual loss of consciousness, and not attended by paralysis, although there may be motor unrest and convulsions; conjugate deviation of the eyes and head, and enforced positions of the body. These are the symptoms that must be con-

trasted with those of cerebral hemorrhage and pachymeningitis hæmorrhagica to make a differential diagnosis from either of these affections—always a difficult and oftentimes an impossible thing to do. The sudden and brusk unrest in cerebral hemorrhage, the early appearance and profundity of the coma, the unilateral paralysis, and frequently the early dissolution, in contrast with the symptoms above, will suggest but may not establish the differental diagnosis between these two diseases. If the cerebral hemorrhage is of the slowly progressive variety, giving rise to symptoms of progressive softening, the differentiation is impossible. It is still more difficult to distinguish it from other forms of non-traumatic meningeal hemorrhage, particularly pachymeningitis interna hæmorrhagica. The fact of the occurrence of the latter disease with degenerative psychoses and neuroses, and that it frequently produces no distinguishable symptoms, contrasted with the diagnostic symptoms that we have mentioned for interpial hemorrhage, may prompt the differentiation.

Treatment.

Treatment is not of much avail, and is in all respects the same as that for cerebral apoplexy. Rest and diminution of blood pressure are the desiderata. The first is to be obtained by keeping the patient in a horizontal position and by attention to the bodily functions of digestion and excretion. The latter is contributed to by the intelligent administration of nitroglycerin and the bromides. A localized intermeningeal hemorrhage in an infant may undergo retrograde changes and become the irritant focus which provokes a Jacksonian epilepsy. In such a case surgical relief should be sought.

HYSTERIA.

BY

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HYSTERIA.

UNDER the term hysteria we distinguish a group of symptoms and of syndromes, some of which are paroxysmal, others more or less permanent; for the latter the name "stigmata" is specially reserved.

HISTORY.

Without entering at great length into the history of this affection, which would be out of place in a work of this nature, we may recall the fact that our knowledge of hysteria goes back to the earliest ages of medicine. It was long considered to be peculiar to the female sex, and its seat was thought to be the uterus. The Hippocratic writers, however, give but a very superficial description of it, and the first account worthy of attention which we have is that of Celsus. It is to be noted, however, that Celsus was not himself a physician, but simply a writer who reflected the opinions of the Greek physicians then resident in Rome. It was a Greek physician also, Galen, who, a century and a half later, was the first to recognize that the affection was not confined to women, but that men might also suffer from its symptoms. But hysteria in the male was not truly recognized until much later, by Lepois and Sydenham. The history of hysteria in the Middle Ages is confounded with that of possession; we find some interesting facts in this regard in the works of Charcot and Richer, "Les démoniaques dans l'art," and "Les malades et les difformes dans l'art;" and in the excellent studies of Richer on hystero-epilepsy. At the time of the Renaissance the womb was still considered to be the seat of hysteria, and Ambroise Paré described the affection under the title "suffocatio matricis."

At the beginning of the seventeenth century Ch. Lepois recognized distinctly that hysteria could occur as well in men as in women—an opinion which was supported sixty and more years later by Sydenham, who understood well the relations between hysteria and hypochondria, and who described very happily a number of the symptoms of hysteria. It is to Mead especially that the honor belongs of having recognized that hysteria is not a disease of any organ, but is a morbid condition of the entire organism: "non unam sedem habet, sed

totius corporis est," he said. But the idea of a uterine localization was not completely eradicated, for we see it coming up again at the beginning of this century in the writings of Louyer Villermay. Georget returned to the doctrine of Ch. Lepois and Sydenham, and understood well the chief differential characteristics of hysteria and epilepsy. Hufeland admitted that hysteria and hypochondria were separated from each other only by sexual differences, but in the same year(1838) Dubois of Amiens again advanced the theory that hysteria was an affection peculiar to women, and he inclined to the belief that its seat was the uterus, an opinion which Landouzy also maintained. Brodie, following Georget and Brachet, returned to the nervous theory and recognized the rôle of the brain and of moral impressions. Briquet returned also to the theories of Sydenham, Lepois, and Georget, and held that the seat of hysteria is in the brain and that it ought to be regarded as a dynamic affection. His book is one of the best works which have ever been published on hysteria, and although we may not find vast generalizations there, we do find in it a number of new facts and an example of good clinical observation.

Ch. Lepois and Sydenham had already remarked that epilepsy may assume an hysterical character during its paroxysm. Sennert had described a uterine epilepsy. Willis had seen hysteria become transformed into epilepsy. There were described later an hysterical epilepsy and an epileptic hysteria (Pomme, Louyer Villermay), and finally a sort of hybrid, hystero-epilepsy, the attacks of which resembled at the same time hysteria and epilepsy. We may see in the same subject epileptiform attacks and then hysteriform attacks, or vice versa, or even so-called mixed crises in which the two neuroses are found together. Charcot has endeavored to show that hystero-epilepsy with independent crises should be regarded as a coincidence of the two diseases in the same individual, while hystero-epilepsy with mixed crises has nothing to do with epilepsy, but is pure hysteria.

Etiology.

Sydenham had already recognized the fact that hysteria is the most frequent of chronic diseases, but its relative frequency is difficult to determine. Up to within a very recent period it was believed that, even if hysteria could occur in men, it was certainly much more common in women. The recent researches of Marie and Souques, however, have shown that in the general hospitals of Paris hysteria is much more frequent among men than among women, although the statistics of Charcot's clinic at the Salpêtrière show that the number of female hysteriacs is twice as great as that of male subjects.[1] If we

hold—and it seems to me not to admit of doubt—that a distinction may be made between the clientèle of the general hospitals, composed exclusively or very nearly so of laborers drawn from the very lowest classes, and that of the Salpêtrière, which includes a greater variety and a greater number of patients drawn from the middle classes, we may conclude that among the lower classes hysteria is most frequent in men, while the proportions are reversed among the higher classes. If these differences may arise under the same surroundings and under the same conditions of heredity, it is an evidence that, besides heredity, we have to consider in the etiology of hysteria certain variable accessory conditions, varying according to the sex and the social surroundings.

Direct and similar heredity is very frequent in hysteria. It was noted by Hoffmann, Georget, Briquet, and Hammond. It has been remarked, in the case of women as well as in that of men, that the transmission occurs more frequently through the mother (Batault). The earlier the hysteria is manifested, that is to say, the more independent it is of external and accidental conditions, the more probably is it hereditary; infantile hysteria is more often hereditary than is hysteria in the adult (Briquet, Peugniez). Homochronism is not very frequent in the heredity of hysteria for the reason that influences capable of acting as exciting causes are many and various.

Although direct and similar heredity is frequent, indirect and dissimilar heredity is still more frequent. Hysteria is a member of the neuropathic group; and we find it associated both in the family and in the individual with neuroses, psychoses, organic diseases of the nervous system, arthritism, tuberculosis (Grasset), and all affections which betray a congenital vice of organization. Hysterical subjects also present rather commonly morphological deviations which show their relation to those of the teratological family; we shall return to this subject later. The frequent association of hysteria with degeneration of all forms enables us to understand how hysteria may have its origin elsewhere than in heredity, just as do the other degenerations. The chronic intoxications and profound disturbances of nutrition from whatever cause may be concerned in the origin of hysteria, and we shall see furthermore that the influences which have been designated exciting causes may have another effect than that of occasional causes.

Just as hysteria belongs to both sexes so may it belong to all ages. It occurs in boys as well as in girls, though less frequently, and it may be seen in children of eighteen months to two years, or possibly even earlier (Chaumier). From the age of seven or eight years it is rather frequent. It is most common in both sexes from the tenth to

the twentieth year, but especially from the fifteenth to the twentieth. It has been seen in men as late as the sixtieth year and in women at a much more advanced period.

Congenital hysteria has not been noted hitherto; what is inherited is a natural defect which is the organic base of the predisposition. This predisposition may require a considerable time for its awakening, and may not awake except as the consequence of violent and repeated shocks. The predisposition is more or less marked, and we may say that it is the greater the earlier it is manifested and the more insignificant is its exciting cause; and inversely it is less as it manifests itself at a more mature age and under the influence of a more intense shock. There are, indeed, certain kinds of shock which may provoke hysterical symptoms in individuals who have no trace of predisposition to the affection; this condition has been called traumatic neurosis, but in reality it differs in scarcely any respect from so-called hereditary hysteria. We see, therefore, that the etiological conditions which have been described under the name of the exciting causes (agents provocateurs)[3] may play the rôle of a predisposing or an exciting cause.

All the conditions which may lead to a depression of the nervous functions and of general nutrition may excite hysteria. The moral causes are equally as efficient as the physical causes, for the simple reason that the emotions are inseparable from certain physical conditions identical with those which are the consequences of traumatic shock.[4] It is not only the lively emotions, such as fear and anger, which are capable of more or less suddenly causing hysteria, but habitual preoccupations are capable of producing the same effect. The exaltation of the imagination developed by a vicious education, religious enthusiasm, beliefs in imaginary powers, the fantastic tales of sorcerers, of ghosts, spirits, and the like, play an important rôle in the production of hysteria and especially of epidemic hysteria. The attempts at hypnotization often cause a moral shock which may be followed by hysterical explosions. The habitual fear which is developed in maltreated children is pregnant with accidents of the same sort. Emotions experienced in dreams may produce the same effects as those in the waking state.[5] Emotions occupy the first rank among the determining causes of hysteria.

The place of traumatism in the etiology of hysteria was for a long time unrecognized. The troubles of the nervous system consecutive to shock, especially to that of a railway accident, are recognized in pathology under the name of railway spine, given to them by Erichsen,[6] by whom they are explained by inflammation of the spinal cord and of its membranes, which inflammation may extend to the enceph-

alic region and then be accompanied by cerebral symptoms. Erichsen's views were accepted by Erb and Leyden, and by physicians in general up to the publication of Page's work,[7] which showed that the brain plays the rôle which had up to that time been attributed to the spinal cord, and that patients in a state of shock are in a mental condition analogous to that of the hypnotized. That hysteria and neurasthenia hold an important place in the pathogenesis of shock is a fact which is shown distinctly by the observations of Page, but which would also be deduced from the observations of Erichsen and even of the older writers. It was Charcot especially who endeavored to show that the nervous symptoms of traumatism were related to hysteria. His views were accepted in America by Walton and Putnam.

In Germany the theory of traumatic lesions of the cord had been abandoned, but Thompsen and Oppenheim attributed the symptoms of shock not to hysteria but to a special neurosis; to traumatic hysteria they added traumatic neurosis.[8] This neurosis was characterized especially, according to them, by the tenacity of the disturbances of sensibility and by a rather cheerful mental condition. Charcot argued with great persistence in favor of the analogy of the troubles seen after traumatism to hysterical symptoms. The fact of curability can hardly, as Oppenheim contended, constitute a characteristic of hysteria. To-day the opinion of Charcot tends to prevail, and it is generally admitted that, as regards its symptomatology, traumatic neurosis does not differ from hysteria.

All forms of shock, whatever may be their nature or intensity, may excite hysteria, and the disturbances caused by an earthquake or by lightning do not necessarily determine graver accidents than a simple contusion received under the most ordinary conditions.

Violent excitations of the organs of special sense may act as traumatisms. I have seen hysterical symptoms follow exposure to the electric light.[9] Sydenham had observed hysteria following a fever which had been treated by venesection and repeated purgation; we here find several debilitating causes, one alone of which would have sufficed. General diseases are often the occasion of hysterical symptoms which may be their first manifestation; such, for example, are pneumonia, pleurisy, acute articular rheumatism, malaria, scarlatina, influenza, diphtheria, and typhoid fever. Zambaco believed in the existence of hysteria symptomatic of syphilis, but the fact is that syphilis, especially secondary syphilis, often occasions an invasion of hysterical troubles, which, however, have no peculiar features. Certain cases of secondary syphilitic anæsthesia have been described, but they are really due to hysteria.

Diabetes may coexist with hysteria or excite it. Chlorosis has

appeared also to be an exciting cause of hysteria, but these two affections really flourish upon the same soil. Chlorosis often coexists with congenital malformations, a fact which would class this affection among the degenerations.

Briquet held that diseases of the genital organs do not predispose to hysteria much more than do any other diseases; nevertheless, in the presence of certain hysterical symptoms, especially ovarian or testicular pain, we may well suppose a morbid condition of the affected organs. As regards the testicle I have often observed that when the gland is painful it was abnormal, either in volume or in position. This, however, does not prove that the causal lesion of hysteria is, even in these cases, in the genital organs; this is well shown by those cases in which ovariotomy has left the hysterical symptoms persistent or even increased, and the appearance of hysteria for the first time after castration proves it still more.

Since the general diseases and the infections may give rise to hysteria, we might also suppose that intoxications would have the same effect, since intoxication plays an important rôle in infection; and this is, indeed, what happens. Works which treat of the nervous accidents of plumbism contain many facts which are referable to hysteria. There is an identity between the sensitivo-sensorial anæsthesias of lead poisoning and those of hysteria. It has been shown that the hemiplegic symptoms of saturnism could be cured or transferred by the magnet and by faradization, just as those of hysteria. It was Charcot who demonstrated the hysterical nature of those phenomena, and since 1886 several writers have related new facts in support of this opinion.

Medical literature contains a number of cases of sensitivo-sensorial hemianæsthesia combined with other manifestations of alcoholism. It was Charcot again who in 1886 showed that many of the so-called hysteroid phenomena occurring in the course of alcoholism belonged really to hysteria, provoked by the intoxication. Other toxic agents may produce the same effects, among them sulphide of carbon (Marie), tobacco (Gilbert), morphine, and opium. It is not only the chronic intoxications which may play the rôle of exciting causes of hysteria, but acute poisoning may have the same result. Thus hysteria has been seen to arise in the course of acute alcoholic intoxication, after the administration of a toxic dose of camphor, and in anæsthesia from chloroform, ether, or nitrous oxide gas.

Hemorrhages and profuse diarrhœa may act as the exciting cause of hysteria, and the same is true of overwork in all its forms. Excessive venery and onanism held an important place among the exciting causes of hysteria; excessive physical labor may have the same

effect, as may also severe mental work. But we must note that intellectual overwork is found especially in men who are preoccupied with their studies or business and in women who are overwhelmed with household cares, while among children the effects which have been attributed to this are distinctly due to a morbid predisposition. Fatigue in any shape may provoke hysteria; and hysteria among men is found often in those who are engaged in the most severe kind of manual labor. Hysteria is frequent also among beggars, prisoners, and the *déclassés* of all orders.

All the above-mentioned determining causes are common to both women and men. In women of certain classes which have been supposed to include the greatest number of hysterical subjects, such as domestic servants and prostitutes, we find united all the conditions of moral and physical depression, such as insufficient or defective alimentation, the intoxications (alcohol), and the infections (syphilis). We see in this an explanation of the fact that hysteria in women is less common in the country than in the city. Late hysteria is more common among women of the leisure classes with whom the imagination is more active and the genital life closes later. Among the poorer classes, where the question of food is always an urgent one, the genital life comes to an end more quickly, and even in those cases in which want and excessive labor are not effective causes. Among the rich, however, sexual excitement is often increased as age advances, especially about the time of the menopause, and then becomes the point of departure for more or less grave hysterical accidents. Coitus reservatus has often an influence on the production of hysteria as well as upon that of other nervous troubles. In general, we may say that the causes of general depression of the organism can act only as exciting causes of hysteria, and that without a predisposition they cannot cause hysterical phenomena.

I have, however, noted several times in the work above mentioned,* that there is a remarkable analogy between the objective phenomena of fatigue and those of hysteria, which we may regard as a chronic fatigue; such are the characters of voluntary contraction, the graphic curve of which presents always the same form, the modifications of general and special sensibility, and of the time of reaction and of association. We may recall also other analogies relating to the perception of subjective sensations—sensations of contrast either simultaneous or successive, delusions, and hallucinations—to the difficulty of isolating the movements, and to the tendency of falling under the domination of exterior physicochemical conditions. In the facts from which these analogies are adduced we find no trace of hereditary or of anterior predisposition. We may recall also that Lowenfeld *

and Pitres [10] have shown that very many of the physical signs of hysteria may be encountered in neurasthenia, acquired through mental or physical fatigue. This analogy of hysterical symptoms with troubles which belong to a morbid condition which may be acquired, or even to a physiological state, enables us to comprehend the possibility of an acquired hysteria.

It is difficult to say whether or not the predisposition to hysteria is more common in certain races and in certain countries; the fact is that the affection is met with in all latitudes and among all races, and may even be seen in certain animals, such as the horse and the dog. A fact worthy of note is that in the less cultured races, where fear, superstition, and a tendency to imitation are seen under their most primitive forms, hysteria is often encountered in epidemics; the savages of our own time reproduce quite faithfully the tumultuous and strange scenes which were frequent in Europe during the Middle Ages and which still occasionally take place in rural districts.

Stigmata.

Writers, even those who do not admit the existence of hysteria without a predisposition having for its base a degeneration, dwell but little upon the morphological characters. I myself am the less inclined to admit of the existence of morphological anomalies among the majority of hysterical subjects, since I am convinced that hysteria may be determined by a violent shock apart from any predisposition. Nevertheless most hysterical subjects do present teratological signs of degeneration. Without doubt hysterical men may present all the external characters of virility, and hysterical women may be remarkable for the development of their specific or accessory sexual characters, nevertheless hysteria, like degeneration in general, tends to level the differences which separate the two sexes, men inclining towards the feminine type and women to the masculine. We may also find in the hysterical all the stigmata of degeneration with this peculiarity, that these stigmata are most frequently predominating on one side, that which is most affected by the sensorial or motor troubles. We shall have to refer to this peculiarity again. These are the morphological anomalies which alone deserve the name of stigmata, they are in effect the only characters which are truly permanent. But it is especially the lasting manifestations, affecting sensibility chiefly, that we designate ordinarily by this name. The functional stigmata of hysteria are rarely permanent, strictly speaking, for they vary in form and in intensity in the course of evolution of the morbid state.

Anæsthesia.

The most frequent of the permanent troubles are those of sensation, namely, anæsthesia or dysæsthesia. Although we often speak of hyperæsthesia, this condition is rather a perversion than an exaggeration of sensibility. Undoubtedly hysterical anæsthesias held the first place among the marks of the devil or the marks of witches, mentioned by demonologists who have described the nervous epidemics of the Middle Ages; but physicians were very dilatory in studying the troubles of sensibility. Sydenham had noted certain facts of dysæsthesia such as rachialgia, and Brodie had also studied several painful syndromes, but it was only towards the end of the first half of the present century that Piorry, Macario, and especially Gendrin, introduced into science the idea of the diffuse or hemiplegic sensitivo-sensorial anæsthesias. This was an important idea, for even if the anæsthesia is not a constant sign, it is one that is nearly always present. Briquet and Lasègue have made important contributions to this study, and they have been followed in our times by numerous observers, especially by Charcot and Pitres. General and special sensations may be affected under all their forms. Pitres has made a classification of the forms of cutaneous anæsthesia which deserves to be retained: 1. Anæsthesia may be total, that is to say, involving all the perceptions, and it may be complete or incomplete; 2. It may be partial, certain sensations only being affected, the others not: (*a*) it is analgesia when there is a loss of the perception of pain with preservation of the tactile sense, (*b*) it is thermo-anæsthesia when the power of perceiving heat is lost, although the tactile and painful sensations are preserved, (*c*) anæsthesia may coexist with thermo-anæsthesia, (*d*) it is electro-anæsthesia when there is a loss only of the perception of electrical sensations, and finally (*e*) anæsthesia may coincide with preservation of electrical sensation only. Analgesia is one of the most frequent troubles of cutaneous sensibility. A remarkable fact, which was noted by Lasègue, is that the patients themselves very rarely are aware of their loss of sensation, and it is only discovered accidentally, as when they have received a burn without perceiving it, or when a prick causes hemorrhage without having excited any sensation. This teaches us that we should study the condition of sensibility with care, that we should look for anæsthesia. In order to study the progress of the anæsthesia we should mark its intensity and distribution by means of diagrams (Richer).

Hysterical analgesia is not only superficial but also involves the nerves, which may be pricked or compressed without result other

than movements in the muscles to which they are distributed. The muscles may also be affected, but a muscle which is insensible to pressure or other stimuli is none the less responsive as regards its motor functions; but the sensation of movement may also be affected as well as that of fatigue (Pitres). The sense of position is lost; with closed eyes the patient is unable to tell the position of his hand, and cannot estimate the amount of energy necessary to maintain his equilibrium, and falls in consequence. Duchenne, of Boulogne, has described paralysis of muscular sense or of motor aptitude independent of sight, which falls into the same category as the preceding phenomena. In this case, as Lasègue has remarked, the touch with the healthy hand may supplement the muscular and visual sensations, warning the patient that the movement has been executed as he desired. The bones, the tendons, and the articulations may also be affected, and one may twist the joints or prick the tendons or the periosteum without exciting pain. Compression of the breasts, of the abdominal viscera, or of the epigastrium (Pitres) may be made without exciting any of the sensations usually aroused by such acts, but certain organs, such as the testicle or the ovary, may give evidence, on the other hand, of an abnormal sensibility, and the same is occasionally true of the mammary glands.

These various troubles of sensibility (anæsthesia, analgesia, thermo-anæsthesia, and electro-anæsthesia) may vary greatly in degree, as may be determined by the different forms of æsthesiometers in use. Not only may the troubles of cutaneous sensibility be wanting, but the anæsthesia, instead of being total, may be dissociated, as noted in the classification of Pitres above mentioned, so that only one of the sensations, that of heat, for example, may be destroyed. Sometimes tactile sensibility alone is preserved, the others being extinguished, hysteria imitating in this the dissociations of syringomyelia.

We sometimes observe in hysterical subjects, following the convulsive paroxysms, a general and total sensitivo-sensorial anæsthesia, which is, however, not permanent. Cutaneous anæsthesia, which is permanent and independent of the paroxysms, is rarely generalized (Briquet), but is more commonly partial, hemiplegic, in disseminated islets or in systematic plaques. The hemiplegic form is a little less frequent than that in islets or plaques. In general, cutaneous hemianæsthesia coincides with a diminished special sensibility on the same side, or exceptionally the anæsthesia and loss of sensibility occupy opposite sides. The hemianæsthesia is on the left side in nearly three-fourths of the cases. Circumscribed anæsthesia appears under the most various forms, and it is this especially which we must search for in examining a case of hysteria. As regards the systematic anæs-

thesia in plaques, we often see it circumscribed to the skin covering or surrounding the organs of sense, the functions of which are weakened or abolished, or limited to a region the muscles of which are paralyzed, whether of the face or of the extremities. The phenomenon of cutaneous anæsthesia superposed upon sensorial anæsthesia is not confined exclusively to cases of hysteria."

In general, as we have remarked, hysterical patients have no knowledge of their anæsthesia, and discover it only by chance, upon the receipt of a burn or a wound which leaves visible evidences, but which has not been felt, or when in suddenly moving from one place to another the impression of the changing temperature is distinctly different on the two sides. The anæsthesia is not accompanied by any sensations of numbness or tingling, and many hysterical subjects who have nocturnal fatigue or paræsthesia with sensations of numbness, cold, or tingling, are nevertheless not led thereby to discover their permanent anæsthesia.

The loss of tactile sensation is accompanied sometimes by a perversion of sensibility, a dysæsthesia, in which the skin cannot bear the least contact or the slightest change of temperature without experiencing pain. This dysæsthesia, which may be unilateral, is accompanied by a diminished activity of the special senses on the same side.

Pitres has described, under the name of aphalgesia, a variety of paræsthesia characterized by the production of an intensely painful sensation through the application to the skin of certain substances which, in a healthy person, would cause only the ordinary sensation of contact; a metallic object, for example, produces a burning sensation. This painful sensation may be manifested on the healthy side as well as on the anæsthetic one.

Hysterical anæsthesia is accompanied by a weakening of certain reflexes. Thus when one tickles the feet of a hemianæsthetic hysterical subject, the reflex occurs exclusively or very much more markedly on the non-anæsthetic side. Tickling of the lateral surfaces of the abdomen may sometimes also provoke only a weak reflex. The sensitive pupillary reflex persists (Pitres), as do also those of the clitoris and of the nipple (Briquet).

Hysterical subjects complain occasionally of a sensation of cold, especially in the extremities on the anæsthetic side, although with the exception of Briquet no one has ever been able to make out a local depression of temperature. Guichon has shown that the subcutaneous absorption of salicylate of sodium takes place equally well on the healthy and on the anæsthetic side; but this fact has no direct relation to the state of the skin itself. It is quite certain that punctured

wounds often bleed less on the anæsthetic than on the healthy side. Pitres explains this fact by assuming a vascular hyperexcitability which is manifested under the influence of traumatism; but the fact is that the puncture becomes surrounded by an urticarial papule analogous to the phenomenon of dermographism. Romain Vigouroux has shown, however, that the electrical resistance is increased on the anæsthetic side; but as we can hardly, in the present state of our knowledge, understand this increased resistance otherwise than in consequence of a diminution of liquids, we may thereby infer that there is a diminished quantity of blood in the member and in the skin. Employing Henocque's method, I have noticed a diminished facility of reduction of oxyhæmoglobin, and the blood withdrawn through a puncture on the anæsthetic side has seemed to me to be richer in corpuscles and in oxyhæmoglobin. All these phenomena, which I should be glad to have demonstrated by other observers, would seem to indicate a diminution in nutritive activity on the affected side. We find the same also in ordinary hemiplegia, especially when it is of long standing.

It is not the skin only which is the seat of hysterical anæsthesia, but we find the same condition in the mucous membranes and in the organs of special sense. The appearance of these different troubles seems to be contemporaneous. The general anæsthesia and that of the special senses are found usually on the same side. Sometimes, however, the eye is more affected on the opposite side, and generally that of the opposite side is affected at least to a certain degree. We may ask whether this apparent exception is not the rule; and indeed the sensibility under all its forms is often notably impaired on the side which is called healthy in hemianæsthetic hysterical subjects; and if we examine them from another point of view we see that the strength of the voluntary movements is diminished on both sides, while the reaction time is increased.

Eyes.—The functional troubles of the eye have a special importance. The existence of hysterical blindness is shown to us in the miraculous cure of the blind during the mystic period of the history of hysteria. The general sensibility of the eye is affected as well as its special sense. The skin of the lids, the conjunctiva, and the cornea itself are devoid of sensation. When the cornea is insensible, while the patient regards fixedly a given object we may bring a cylinder of paper in contact with the cornea without producing any effect, the lids and the eye itself remaining motionless as long as the foreign body is not brought within the field of vision. The oculo-pupillary reflex is obtained as promptly as in the normal state, if, instead of bringing the paper in contact with the conjunctiva, we bring it a certain distance in front of the pupil; the reflex is then determined

through stimulation of the retina, which may have lost the perception of colors, but which still distinguishes light from darkness. There is in general a relation between the intensity and extent of the anæsthesia of the integuments and the diminished special sense. Hemianæsthetic hysterical subjects who have no narrowing of the visual field nor achromatopsia preserve the sensibility of the conjunctiva; those who have lost the power of perception of one or several colors and have a more or less marked narrowing of the visual field have also anæsthesia of the conjunctiva. Those who have complete achromatopsia with almost no visual field have lost not only the conjunctival sensibility but also that of the cornea.[12] However, as Pitres has remarked, the lacrymal reflex is not ordinarily lost and is excited just as in the normal condition by contact with the conjunctiva.[13]

Walton and Gradenigo have confirmed my observations in regard to the relation between anæsthesia of the integument and the loss of special sense; they have shown that anæsthesia of the external auditory canal is the more marked as deafness is the more pronounced.[14]

One of the most important signs of visual anæsthesia, according to Charcot, and perhaps the most frequent, according to Dana, is the narrowing of the visual field. This narrowing may be roughly appreciated by having the patient look steadily at the nose of the observer who moves his finger in all directions towards the point of fixation; but the campimetre is necessary for a regular examination, the results of which may be registered on the charts which are in ordinary use in most clinics. The normal visual field is not circular, but extends farther outwards and downwards, while the inner and upper borders are less projecting. As a rule the narrowed visual field preserves the normal shape, that is, the contraction is concentric. However, the regularity of the visual field can be determined only when the examination is made rapidly and while the patient is alone with the observer; for hysterical subjects have very rapid modifications of sensibility under slight influences, such as a noise, a change in the light, etc., and all these influences may cause apparent irregularities in the visual field. We may say that ninety times out of a hundred the narrowing of the field of vision is bilateral, as are all the other visual troubles, but as a rule one is more narrowed than the other, and the side of the greatest contraction is ordinarily that affected with the hemianæsthesia. Total blindness is exceptional, and when it occurs is ordinarily temporary only, occurring after a convulsive paroxysm or under other accidental conditions. Freud believed in 1893, as I had already held ten years before, that hysterical hemiopia had not been seen and that its occurrence was not probable; and the cases re-

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The coexistence of hysteria with a great number of affections of e nervous system explains how it is that the visual troubles of hys-ria have been attributed to these various maladies. We should te, also, that most of the troubles of vision in hysterical subjects, d especially the modifications of the visual field, may be provoked all the conditions which lead to a sudden depression of the nervous stem, even by fatigue; and further, that in hysteria the visual de-cts present a considerable variability. The concentric narrowing of e visual field, the dyschromatopsia, and the diminished acuity of sion are frequently manifested after an epileptic attack. I have en found in these cases, just as in hysteria, the superposition of gumentary anæsthesia. After Oppenheim and Thompsen and ichon I also have observed in epileptics permanent troubles of sion, especially narrowing of the visual field. Although, therefore, e may say that these troubles are more marked and more variable hysteria, we cannot regard them as absolutely pathognomonic of is morbid state.

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movements, measured by the dynamometer, increases considerably in the affected member. If the stimulus is applied to a limb of the healthy side we see anæsthesia appear at the point of application of the æsthesiogenic substance, and spread from this as a centre. Whichever may be the side on which the application is made, we see the passage of the anæsthesia from one side to the other; this is the phenomenon of *transference*, which is constituted by a series of oscillations or of passages of insensibility from one side to the other. Following these oscillations the hemianæsthesia may disappear or at least remain less pronounced. Sometimes the hemianæsthesia remains stationary for a time on the side opposite to that in which it was primarily, or it may remain on the same side on which it was before without any appreciable change. The duration of the consecutive oscillations may vary from a few minutes to an hour or even more.

A certain number of agents which cause a momentary general stimulation may provoke a transference just like those which act locally; such are static electricity and cold or hot water. Subcutaneous injections of pilocarpine or inhalations of nitrite of amyl, ether, or chloroform may provoke a transference, consecutive oscillations, and a return of sensation. In registering by means of the dynamograph the variations of energy of voluntary movements occurring in transference produced by the application of a magnet to the normal side, I have remarked that there is first a certain increase of strength on this side.

All æsthesiogenic agents have not the same influence over a given patient. Burq has very well noted the differences in metallic sensibility, and it is the same in the case of the other æsthesiogenic agents, several of which may have no effect on a certain patient.

The physiology of the æsthesiogenics is little understood. Their action has been referred to the imagination or to electrical currents, but the fact is that we are still in ignorance as to them. Certain painful phenomena may be cured by the application of revulsives to the opposite side. The same means may provoke a transference of the epileptic aura. Even in healthy subjects similar effects have been noted. Hoppe, for example, has observed that if the thumb or index finger is firmly pinched, a notable diminution of sensibility is caused in the corresponding parts on the opposite side, and other observers have reported facts of the same nature with which also we may compare synkinesis and the synalgesias. The transference may be obtained also in hemianæsthesia with organic base (Vulpian, Bourceret, Grasset), so that the hemianæsthesia due to lesion of the posterior third of the internal capsule, which differs in no way from hysterical anæsthesia, even in the character of the visual disturbances, cannot

through stimulation of the retina, which may have lost the perception of colors, but which still distinguishes light from darkness. There is in general a relation between the intensity and extent of the anæsthesia of the integuments and the diminished special sense. Hemianæsthetic hysterical subjects who have no narrowing of the visual field nor achromatopsia preserve the sensibility of the conjunctiva; those who have lost the power of perception of one or several colors and have a more or less marked narrowing of the visual field have also anæsthesia of the conjunctiva. Those who have complete achromatopsia with almost no visual field have lost not only the conjunctival sensibility but also that of the cornea.[12] However, as Pitres has remarked, the lacrymal reflex is not ordinarily lost and is excited just as in the normal condition by contact with the conjunctiva.[13]

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ported by Janet in the *Archives de Neurologie* in 1895, and by Lannois and Tournier in the *Revue de Médecine* in 1896, are not of such a character as would convert one to the contrary opinion.

The narrowing of the visual field regards in general both vision for colors and that for white light. In the normal state the visual field for white light is the most extensive, and after that come, in the order of their extent, blue, yellow, orange, red, green, and violet, the field for which is the narrowest of all. In hysteria the field for colors is contracted concentrically in the physiological order, that is to say, the field for violet may be so narrowed that this color is no longer perceptible, and the fields for the other colors disappear successively. It is the rule, however, that the field for red is narrowed the least and disappears the last (Charcot, Parinaud). Sometimes even the field for red is more extensive than that for white light (Parinaud). Sometimes the vision for colors disappears completely while that for form persists, and the patients perceive objects as of a gray or dirty white color only. It is not excessively rare to see the vision for colors disappear in the two eyes in a different order. In general the field for colors is more affected on the hemianæsthetic side, but it is as changeable as are the other modifications of sensibility, being often more marked after a paroxysmal attack. Rarely the narrowing of the visual field coincides with a central scotoma (Parinaud).

It may be that the acuteness of vision is affected in the same measure as the visual field, but this is the exception, and in general the acuteness of the sight is but little affected.

Hysterical amblyopia is often accompanied by diplopia or monocular polyopia (Parinaud). A pencil or other object is placed near the eye and gradually drawn away from it. At first it is seen single, but at a distance of from ten to fifteen centimetres a second image appears, usually on the temporal side. As the object is made to recede from the eye the two images separate from each other, and not very uncommonly a third, less intense, image appears on the opposite side. If the object is still farther removed the images become blurred and are no longer seen distinctly. We may observe at the same time very marked macropsia or micropsia upon moving the object away from or towards the eye (Parinaud).

In unilateral hysterical amaurosis, the eye which does not see when the other is covered may see in binocular vision and especially in stereoscopic vision. The reëstablishment of sight in the amblyopic eye at the moment of binocular vision has regard only to central vision, for the visual field still remains narrowed (Parinaud).

If we revolve before an hysterical subject a disc on which are painted complementary colors, the perception of one of which at least

has been lost by the patient, he is able to perceive the compound color. This experiment of Regnard shows that the loss of vision is of central origin. The fact noted by Parinaud of the reëstablishment in binocular vision of central vision with persistence of the narrowing of the visual field in the amaurotic eye indicates that there are separate cerebral centres for peripheral and for central vision, and that in central vision each eye is in relation with the two hemispheres. The experiments of Pitres with the Flees box point in the same direction. The relation of each eye with the two hemispheres in central vision is supported also by facts of an entirely different order.[14, 15]

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the same side. We should avoid the use of the term hyperæsthesia, which is very inexact.

Variations in Intensity.—The anæsthesia is in general variable in its intensity, but it may preserve the same distribution for years, even for a long time after the other hysterical symptoms have disappeared. We have seen at the Salpêtrière two hysterical patients whose hemianæsthesia had persisted for more than forty years. Some form of anæsthesia persisting up to old age is seldom absent in the adult, especially in man, but it is often wanting in the child. Local anæsthesias may present the same persistence, especially when they are in relation with local troubles of another form, such as paralysis, contracture, etc. The anæsthesia seems to vary spontaneously, so slight and ephemeral may be the influences which provoke these variations; all physical agents may exert an action on it. It is often more marked in the morning when the patient awakes; it is influenced not only by darkness but also by sleep and inaction. The stimuli of sight, hearing, touch, smell, or taste may modify it in various ways; moderate stimuli diminish it, very intense or painful ones may increase it. The influence of stimuli applied to the skin was recognized by Duchenne, of Boulogne, and by Briquet, who noted that faradic electrization restores the sensibility rapidly over a more or less extensive area surrounding the point to which the stimulus has been applied. The organs of special sense and their mucous coverings may participate in this restoration, but amblyopia often proves refractory, as does also muscular anæsthesia. Other cutaneous stimuli may have the same effect, as for example vesication, friction, the application of croton oil, sinapisms, metallic plates, magnets, and mechanical vibrations. All these agents have been grouped under the name of *æsthesiogenic*. A committee appointed by the Société de Biologie of Paris, composed of Charcot, Luys, and Dumontpallier, to whom were added Gellé, Landolt, and Regnard, undertook the study of these agents, and it was in the course of these experiments that Gellé discovered by means of his binauricular tube the phenomenon of transference, and that Charcot studied the consecutive oscillations.

The stimulus restores the sensibility first over a limited area, and little by little the sensible zone extends until sometimes it embraces the entire hemianæsthetic side; then the anæsthesia reappears in patches in the parts in which sensation had been restored the most recently (the return anæsthesia of Burq). This phenomenon, which may last for half a minute to nearly an hour, is accompanied by other no less interesting phenomena. For example, pricks which were bloodless on the anæsthetic skin begin to exude blood, the temperature rises as the sensibility returns, and the strength of voluntary

movements, measured by the dynamometer, increases considerably in the affected member. If the stimulus is applied to a limb of the healthy side we see anæsthesia appear at the point of application of the æsthesiogenic substance, and spread from this as a centre. Whichever may be the side on which the application is made, we see the passage of the anæsthesia from one side to the other; this is the phenomenon of *transference*, which is constituted by a series of oscillations or of passages of insensibility from one side to the other. Following these oscillations the hemianæsthesia may disappear or at least remain less pronounced. Sometimes the hemianæsthesia remains stationary for a time on the side opposite to that in which it was primarily, or it may remain on the same side on which it was before without any appreciable change. The duration of the consecutive oscillations may vary from a few minutes to an hour or even more.

A certain number of agents which cause a momentary general stimulation may provoke a transference just like those which act locally; such are static electricity and cold or hot water. Subcutaneous injections of pilocarpine or inhalations of nitrite of amyl, ether, or chloroform may provoke a transference, consecutive oscillations, and a return of sensation. In registering by means of the dynamograph the variations of energy of voluntary movements occurring in transference produced by the application of a magnet to the normal side, I have remarked that there is first a certain increase of strength on this side.

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The physiology of the æsthesiogenics is little understood. Their action has been referred to the imagination or to electrical currents, but the fact is that we are still in ignorance as to them. Certain painful phenomena may be cured by the application of revulsives to the opposite side. The same means may provoke a transference of the epileptic aura. Even in healthy subjects similar effects have been noted. Hoppe, for example, has observed that if the thumb or index finger is firmly pinched, a notable diminution of sensibility is caused in the corresponding parts on the opposite side, and other observers have reported facts of the same nature with which also we may compare synkinesis and the synalgesias. The transference may be obtained also in hemianæsthesia with organic base (Vulpian, Bourceret, Grasset), so that the hemianæsthesia due to lesion of the posterior third of the internal capsule, which differs in no way from hysterical anæsthesia, even in the character of the visual disturbances, cannot

and in cases in which I have observed it, it has been coincident with an anæsthesia of the special senses. We must not confound hypnotic hyperexcitability with sensory hyperæsthesia properly speaking. Dysæsthesia distributed in systematic regions is generally accompanied, like anæsthesia, with motor troubles, and especially with contractures or spasms. As a rule the dysæsthesia continues for some time after the other troubles, the muscular contractures or arthralgias, have disappeared.

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Hysterical arthralgia may be seen in men, although it is more common in women; it may also be seen in children. It may be the first symptom of which an hysterical patient complains. But even when pain is the first symptom, the contracture of the muscles which move the affected articulation is not slow to appear and disables the member, and it is rare that the pain remains as the only symptom. The pain is increased by pressure and movement; it is rarely limited to one point, but extends throughout the whole limb; and pinching of the skin is usually more painful than pressure on the joint. As a rule the cutaneous pain is more marked in a segment surrounding the

morbid sensibility so that the least touch will excite a paroxysm. Ocular hysterogenic points are, however, rare (Lichtwitz).

In the *mouth* general sensibility is usually abolished, but von Rabenau claims to have seen tactile sensibility alone preserved. According to Lichtwitz galvanic sensibility is generally diminished, but this diminution would seem to have no relation with the other forms of sensibility.[18]

Taste is also affected at the same time as the sensibility of the buccal mucous membrane. In order to appreciate this form of anæsthesia we place at the posterior portion of the tongue, on each side separately, solutions of certain crystallized sapid substances of known strength. As a rule gustatory anæsthesia is unilateral if the general anæsthesia is unilateral, but it may be general or wanting when there is a unilateral loss of general sensibility of the mucous membrane. When gustatory anæsthesia exists, general anæsthesia is never wanting (Henrot). The gustatory field may be narrowed and restricted to the posterior part (Lichtwitz). The field of electrical sensation is also diminished, and sometimes this sensation is wholly abolished. The disturbances of taste, which render all food insipid, play an important rôle in the tendency which all hysterical subjects have to prefer highly seasoned food or substances which have a peculiar taste or odor. They are also of importance in the etiology of anorexia, although the reflex salivary secretion does not appear to be affected.

Chairon speaks of anæsthesia of the epiglottis as a pathognomonic sign of hysteria; but anæsthesia of the larynx, which is usually bilateral, even when the skin is affected on one side only, is frequently wanting and may be encountered in individuals who are in no sense hysterical (Gougenheim).

Nose.—The nasal mucous membrane is, according to Lichtwitz, the least frequently affected in general anæsthesia. Nevertheless, most observers state that it follows the usual law. We must study changes in special sensation by Passy's method, that is to say, by the use of solutions in definite proportions of crystallized odoriferous substances, evaporating in a vessel of given capacity. The loss of smell spares the side which the general anæsthesia spares, from which it results that the patients are wholly ignorant of their loss of sensation.

Ears.—Deafness always manifests itself most markedly on the hemianæsthetic side. This deafness usually varies in intensity in proportion to the general anæsthesia. Deafness for sounds transmitted by the cranial bones is greater than that for sounds transmitted by the air (Walton). There is at the same time a narrowing of the auditory field and the power of appreciating direction is weakened.

of contact, and the bones may possibly undergo a certain degree of fatty degeneration. It sometimes happens that a rheumatic or tuberculous arthritis become engrafted on an hysterical arthralgia. The joint, which was already probably a point of lessened resistance, since it had been selected by the neurosis, has now become so without question in consequence of the apparent or real trophic disturbance, and the localization of the secondary affection follows as in a certain sense a necessary consequence. In other cases an hysterical affection in one joint is associated with an organic one in another. Thus, in a tuberculous subject already suffering from an organic affection of the foot, there may develop, under the influence of a previously existing neuropathic condition, various painful contractures which involve the muscles of the hip and give rise to a diagnosis of coxalgia; but all the symptoms of such an affection disappear as soon as the patient is brought under the influence of chloroform. The disappearance of an hysterical arthralgia may be sudden or gradual. The affection may subside suddenly, but more commonly it disappears gradually. The muscles and tendons may be painful. Myodynia, often connected with rigidity, is more frequent. Achillodynia is perhaps the only form of painful tendon."

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It is to Charcot that we owe the discovery of the hysterogenic zones. In 1873 he described the ovarian dysæsthesia of the hysterical and showed that the hysterical attack was often preceded by an aura which started from the ovarian region; and that, in certain patients, pressure in this region may provoke an attack or arrest one. Following this he discovered other regions in which the same peculiarities were to be observed. Although we find in the history of epidemic possession some striking examples of hysterogenic zones (Gilles de la Tourette), and also a certain number of isolated cases in the writings of others (Mercado, Willis, Boerhaave, Brodie), nevertheless the systematic study of the subject dates from recent times.

The hysterogenic zone is a circumscribed area, painful or not, which is the seat of special sensations, constituting the prodromes of the attack, and compression of which may provoke or arrest the paroxysmal phenomena, either wholly or partially. This definition indicates that the hysterogenic zone may indifferently either provoke

the same side. We should avoid the use of the term hyperæsthesia, which is very inexact.

Variations in Intensity.—The anæsthesia is in general variable in its intensity, but it may preserve the same distribution for years, even for a long time after the other hysterical symptoms have disappeared. We have seen at the Salpêtrière two hysterical patients whose hemianæsthesia had persisted for more than forty years. Some form of anæsthesia persisting up to old age is seldom absent in the adult, especially in man, but it is often wanting in the child. Local anæsthesias may present the same persistence, especially when they are in relation with local troubles of another form, such as paralysis, contracture, etc. The anæsthesia seems to vary spontaneously, so slight and ephemeral may be the influences which provoke these variations; all physical agents may exert an action on it. It is often more marked in the morning when the patient awakes; it is influenced not only by darkness but also by sleep and inaction. The stimuli of sight, hearing, touch, smell, or taste may modify it in various ways; moderate stimuli diminish it, very intense or painful ones may increase it. The influence of stimuli applied to the skin was recognized by Duchenne, of Boulogne, and by Briquet, who noted that faradic electrization restores the sensibility rapidly over a more or less extensive area surrounding the point to which the stimulus has been applied. The organs of special sense and their mucous coverings may participate in this restoration, but amblyopia often proves refractory, as does also muscular anæsthesia. Other cutaneous stimuli may have the same effect, as for example vesication, friction, the application of croton oil, sinapisms, metallic plates, magnets, and mechanical vibrations. All these agents have been grouped under the name of *æsthesiogenic*. A committee appointed by the Société de Biologie of Paris, composed of Charcot, Luys, and Dumontpallier, to whom were added Gellé, Landolt, and Regnard, undertook the study of these agents, and it was in the course of these experiments that Gellé discovered by means of his binauricular tube the phenomenon of transference, and that Charcot studied the consecutive oscillations.

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be distinguished by this phenomenon either. Neither can the absence of cerebral lesion be deduced from the absence of paralysis of the inferior facial, for this symptom may be wanting in case of the organic lesion in question, and one finds it usually, the contrary opinion notwithstanding, in a certain degree in hysteria.

Charcot has shown that the anæsthesias of syringomyelia may have a distribution on the surface of the body like that of hysterical anæsthesia, in limited geometrical zones, in segments of the limbs, under the hemiplegic form, etc. On the other hand, we may see the syringomyelic dissociation of sensibility in hysteria. If to the clinical picture of syringomyelia we add the narrowing of the visual field, which Déjerine and Tuilant have included among its symptoms, we shall see that syringomyelia may present disturbances of sensation very similar to those observed in hysteria; but a diagnosis may easily be made from the symptomatic associations.

Abolition is not the only disturbance of sensation in hysteria, for instead of being abolished this may often be perverted, so that the most normal stimuli may excite pain, and certain functions which in the normal condition are performed without giving rise to any sensation are accompanied by pain, and certain organs or certain points on the body become the seat of constant pain. Sydenham recognized certain of these forms of pain as spinal hyperæsthesia and the clavus hystericus; after him came Brodie, then Beau, Schutzenberger, and Briquet; and finally Charcot, who studied especially the painful points regarded as hysterogenic zones.

Dysæsthesia.

Dysæsthesia or painful sensibility may affect the same parts as anæsthesia, and like the latter also may affect all the modes of sensation. The pain may be provoked by simple contact, by pricking, by cold or heat. The dysæsthesia manifests itself under different forms; sometimes the patients complain of a sensation of burning, of tingling, of stinging, of numbness or of heat, and sometimes they describe their sensations with a richness of extraordinary metaphorical expressions which may assume a truly delirious character. For example, a lady assured me that the sensation of burning which she felt on the right side was so far from being imaginary that after she had opened a door and her maid took hold of the handle a short time after, she was forced to let go of it at once because it burned her.

Cutaneous dysæsthesia may present the same variety of distribution as unilateral anæsthesia, in disseminated islets or in systematic areas; it is rarely general. It may manifest itself spontaneously or may be provoked by external stimuli. Hemidysæsthesia is rare,

and in cases in which I have observed it, it has been coincident with an anæsthesia of the special senses. We must not confound hypnotic hyperexcitability with sensory hyperæsthesia properly speaking. Dysæsthesia distributed in systematic regions is generally accompanied, like anæsthesia, with motor troubles, and especially with contractures or spasms. As a rule the dysæsthesia continues for some time after the other troubles, the muscular contractures or arthralgias, have disappeared.

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affected articulation and in the part above. When the muscles are contractured the integument covering them often becomes itself painful. Sometimes the dysæsthetic zone becomes so irritable that the least contact provokes a convulsive attack. In hysterical coxalgia there may be pain in the knee just as in true tuberculosis of the hip, but, as a rule, the nocturnal pains are absent in the hysterical; furthermore, distraction singularly moderates the pain, which is, on the contrary, increased by the simple attempt at examination. In hysterical coxalgia the attitude of the patient and the deformity may simulate exactly organic hip disease; but the contracture is often exaggerated, involving almost the entire member or, at any rate, muscles which have nothing to do with the painful articulation. The limp, which is only exceptionally noted, since the contracture is so excessive that the patient refuses all attempts to walk, is often irregular, intermittent, and choreiform.

Whatever may be the articulation affected, there are in general no other symptoms than pain, contracture, and loss of power; but sometimes there is observed a swelling, which has no resemblance to that accompanying suppuration, and which must be attributed to œdema. It may happen also that the hysterical arthralgia is accompanied by muscular atrophy presenting the characters of the atrophy following organic affections of the articulations, the reaction of degeneration, and fibrillary tremors (Charcot and Babinsky). This muscular atrophy may be accompanied by a lowering of temperature in the affected member. Although muscular atrophy is less common in hysterical than in organic arthralgias, it is apt to be more extensive in the former; instead of attacking exclusively the extensor muscles of the joint—the gluteal muscles in the case of hip disease, the triceps femoris in that of arthritis of the knee—it involves often the muscles below and those having no functional relation with the painful articulation. In several cases of hysterical arthralgia, in which amputation has been performed, the articulation has been found perfectly sound. When the patient is examined under chloroform narcosis, also, it is found, as soon as relaxation of the contractured muscles is complete, that the movements of the joint are perfectly free. As the effect of the anæsthetic wears away, the stiffness returns before the pain, and the dysæsthesia is reëstablished in the superficial parts before it is in the deeper ones. Finally, as the affection disappears, the cutaneous dysæsthesia remains longest, and until this has entirely disappeared our prognosis should be reserved.

Under the influence of prolonged immobility hysterical arthralgia may result in the formation of fibrinous adhesions, just as do contractures of long standing, the cartilages may become eroded at the points

of contact, and the bones may possibly undergo a certain degree of fatty degeneration. It sometimes happens that a rheumatic or tuberculous arthritis become engrafted on an hysterical arthralgia. The joint, which was already probably a point of lessened resistance, since it had been selected by the neurosis, has now become so without question in consequence of the apparent or real trophic disturbance, and the localization of the secondary affection follows as in a certain sense a necessary consequence. In other cases an hysterical affection in one joint is associated with an organic one in another. Thus, in a tuberculous subject already suffering from an organic affection of the foot, there may develop, under the influence of a previously existing neuropathic condition, various painful contractures which involve the muscles of the hip and give rise to a diagnosis of coxalgia; but all the symptoms of such an affection disappear as soon as the patient is brought under the influence of chloroform. The disappearance of an hysterical arthralgia may be sudden or gradual. The affection may subside suddenly, but more commonly it disappears gradually. The muscles and tendons may be painful. Myodynia, often connected with rigidity, is more frequent. Achillodynia is perhaps the only form of painful tendon.[88]

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or arrest the spasmodic paroxysms, but rather commonly a zone endowed with special irritability has only the provocative or the suspensory property, and not both. Sometimes a zone is provocative on superficial pressure and suspensory on deep and strong pressure (Pitres). These zones are either tegumentary or deep; the tegumentary zones may be found on the mucous membranes as well as on the skin; the deep zones are most commonly visceral (ovary, testicle, mamma). These hysterogenic zones are not always spontaneously painful, and sometimes they are discovered only as a result of a most careful examination, which in itself may be the occasion of the first convulsive attack. They may be single or multiple. In general we are unable to discover the cause of their localization, but I have several times noted that a painful testicle in an hysterical man is an abnormal one. The head and trunk are the seat of predilection for these zones, but they are also seen on the extremities, especially in the articular folds. Their extent varies, we may say, infinitely; sometimes they are limited to a spot which may be covered by the tip of the finger, at other times they occupy an extended area. The special or neuralgic sensibility of these zones is very variable and is often increased upon the approach of a paroxysm. The hysterogenic zones may be situated in the midst of an anæsthetic patch, and in general in hemianæsthetics they are found on the same side as the anæsthesia. As a rule there is no visible change in these areas, although Buet has observed a loss of the hair over the hysterogenic zones on the scalp. When the zones are spontaneously painful the patients often assume special attitudes to protect them from all contact; but it is not only by pressure that the hysterogenic properties of these zones are manifested, for cold or heat may produce the same effect (Gaube).

Under certain conditions the hysterogenic zones may disappear, change their position, or lose their property in part, as is sometimes seen, for example, after a paroxysm. The influences which may cause a change of location are those which may act simultaneously upon all the zones of the body or only upon those in a special part. Among the general measures are static electricity, galvanization of the nerve centres, and inhalations of anæsthetic substances. The local means are local anæmia such as that which results from the application of an Esmarch bandage, refrigeration by an ether spray and the like, sinapisms, hypodermic injections even of simple water, local faradization or galvanization. In the case of cutaneous zones, especially if they are spontaneously very painful, direct applications may excite an hysterical paroxysm, and the stimuli, especially the electrical ones, should be applied at the periphery of the sensitive zone.

Some of these dysæsthetic or hysterogenic zones deserve a special

description by reason of their peculiar character. The *clavus hystericus*, well known to Sydenham, is one of the most frequent painful phenomena in hysterical subjects, being found in six cases out of seven, according to Briquet. It may have its seat on any part of the head, but most commonly it is found in the temporal regions or over the sinciput; its area is very limited. The patients compare the pain which characterizes it to that which would be caused by the driving of a nail into the skull, or by the application of a hot coal or of a piece of ice. The pain may be superficial or may appear to be seated deeply in the bone or in the brain itself; it is sometimes relieved by pressure. It is continuous in character and may last from a few days to several weeks. Briquet says that it is frequently accompanied by chills, vomiting, digestive troubles, and sometimes by fever. It is in the category of cases in which the latter symptoms occur that the meningo-encephalic (Saint-Ange) and meningitic (Arnozan) forms of hysteria belong. These forms, which are seen especially in women, may also be encountered in children (Sollier, Ollivier). The onset may be brusk or it may be preceded by prodromes resembling in every respect those of tuberculous meningitis; and the later symptoms, such as violent cephalalgia, vomiting, diplopia, delirium, prostration, the so-called *tache méningitique*, and obstinate constipation, all tend to keep up the simulation. The knowledge of the patient's antecedents, the coexistence of troubles of sensibility, and other signs of hysteria, and especially of dysæsthetic zones of the scalp, together with the absence of fever, will render the diagnosis of hysteria easy. The liability to error is much greater when fever is present, yet it would seem that the fever, when present in pseudomeningitic hysteria, is always due to some other cause, such as angina, vaginitis, etc., for which we must search carefully (Gilles de la Tourette).

Among the painful stigmata have been ranged certain phenomena which ought rather to be included among the painful paroxysms. For example, we may mention ophthalmic migraine which is not rare among hysterical subjects, and which is sometimes so closely associated with the other hysterical symptoms that it appears to form part of the symptom complex (Babinsky). We also sometimes see paroxysmal facial neuralgia which presents the characteristics of hysterical manifestations and which is accompanied rather frequently by systematic dysæsthesia of the skin and mucous membrane. Sydenham recognized hysterical odontalgia, and several writers have also reported cases of facial neuralgia. Charcot noted the vesperal character of hysterical neuralgia, comparing it with the matutinal character of ordinary tic douloureux. Briquet found that compression frequently relieved the pains in the head, and I have often made use of a com-

pressor with metallic spring, which I have found serviceable, and also of a leaden cap.

Hysterical *rachialgia*, which was recognized by Sydenham and Brodie, has been made the subject of a careful study by Briquet. It is very common and there are few hysterical patients who have never experienced pain in the back. It is seated along the line of the spinal apophyses and hardly ever passes beyond the vertebral grooves; it may extend along nearly the entire length of the dorsal spine, but more commonly it is limited to two, three, or four vertebræ. Sometimes the pain is deep-seated only, but as a rule there is also an exquisite sensibility of the skin, the least touch of which is the signal for a violent reflex explosion. Usually the significance of this pain is well recognized by the patient who assumes more or less strange attitudes, in order to avoid any contact which might excite attack. Briquet noted the hysterogenic property of these rachialgic points. Alongside of these spinal pains which have the permanent characters of stigmata, we have to note other dysæsthesias of the thoracic walls, such as thoracalgia and pleuralgia, the general character of which is that the pain is seated in the skin; and by this we are able to distinguish them from the pains due to affections of the cord, which they strongly resemble. Motor trouble, paralysis or contracture, may be added to the sensory affections, but, as Brodie remarked, it is not that the muscles do not obey the will, it is that the will does not act. Sometimes the motor and sensory troubles are accompanied with an apparent deformity of the spine (P. Richer). In children the different troubles may be mistaken for manifestations of vertebral rachitis (Grancher). In all these cases a search for other stigmata of hysteria is very important from a diagnostic point of view. We often see associated with rachialgia a pleurodynia which may sometimes have the character of a girdle pain, and which may make us the more think of locomotor ataxia since the latter affection may coexist with hysteria. These thoracic pains may also be associated with pain in the epigastrium, gastralgia or hepatalgia, which may present exacerbations from time to time under the form of crises resembling the visceral crises of locomotor ataxia.

Hysterical *angina pectoris* is a form of thoracalgia. It does not differ in its symptoms from true angina, and it may coexist with organic heart disease (Landouzy). What characterizes it especially is its early appearance, for it may be seen in childhood and almost always in persons under forty years of age; another distinctive sign is its frequency in those of the female sex. Angina pectoris may be the first manifestation of hysteria; it often presents a remarkable obstinacy, and the attacks sometimes recur at very frequent intervals.

The attack is often sudden in its onset and occurs at night, in which the affection differs from true angina as usually observed. There are often found at the same time more or less extensive dysæsthetic zones of the skin, and especially rachialgia. The increase of sensibility of these zones often marks the beginning of an attack, the pain of which reaches its maximum almost immediately. The skin of the precordial region is the seat of a smarting sensation, and the left side is as if compressed in a vice; the pains radiate into the neck and upper extremity, chiefly along the course of the cubital nerve, and sometimes throughout the entire left side of the body. In exceptional cases the pain commences in the little finger to the left side, or in the toes of the left foot, and thence spreads to the trunk. In almost all cases the skin, whatever may have been the mode of onset, is affected with a very distressing dysæsthesia. In some cases we note very marked vasomotor troubles, the skin of the face passing through all shades of color, livid, blue, and red. Sometimes the same phenomena are manifested in the left arm, or the entire left side becomes cold, and local asphyxia of the fingers is produced. During the height of the attack we often find palpitation, intermittent heart beats, sometimes a scarcely perceptible and often very rapid pulse. The respiration is also disturbed, and we may notice irregularity, dyspnœa, oppression, or complete arrest.[17] Compression of the phrenic nerve at the level of the scaleni is often painful, as is sometimes that of the cervical sympathetic. The attack is accompanied ordinarily by an extremely distressing sense of anxiety with a tendency to syncope. The severity of the attacks varies considerably; they are sometimes very light and very brief or they may last for several hours; in the latter case we have to do with subintrant attacks. The exciting causes of a paroxysm are often exceedingly slight, sometimes a dream may cause it, or it may be excited by pressure on an hysterogenic zone (Rendu).

In the abdominal region there are dysæsthetic and often hysterogenic zones which are analogous in their seat in the two sexes; thus the hypogastric zones correspond in women to the ovary, and in man to the prolongation of the spermatic cord. These zones quite frequently extend to the skin of the external genital organs, such as the labia majora or the scrotum. The last-named parts may be alone affected. The hypogastric and genital cutaneous zones are generally unilateral, but they may be found on both sides.

We may finally mention *coccygodynia*, a dysæsthesia seated at the lower extremity of the coccyx and affecting both skin and deeper tissues. This is a very lasting condition and may exist alone.

Dysæsthesia of the Mucous Membranes.—Rosenthal had remarked that we could excite hysterical attacks by touching with a speculum

the cervix uteri, but it is to Lichtwitz that we are indebted for a study of the dysæsthesias, especially the spasmogenic dysæsthesias of the mucous membranes, which exist usually in the form of more or less limited zones. He studied them on the mucous membranes of the nose, the mouth, velum palati, pharynx, larynx, Eustachian tubes, cornea, conjunctiva, and lacrymal ducts. These mucous zones often correspond to the cutaneous zones; they are sometimes unilateral, sometimes symmetrically situated on the two sides. An interesting fact is that noted by Lichtwitz that the points affected with spasmogenic dysæsthesia are often the seat of anatomical lesions. Contact with the dysæsthetic mucous membranes excites unexpected reactions during an examination of the larynx, of the ear, of the Eustachian tube, etc., recalling laryngeal vertigo or Ménière's vertigo. The vaginal mucous membrane may be the seat of a dysæsthesia, contact with which may provoke either local spasm, vaginismus, or generalized attacks. Gilles de la Tourette has noted a spasmogenic dysæsthesia of the urethra; the anal mucous membrane may also be affected and give rise to local spasms analogous to those provoked by fissure of the anus, as I have seen in one case. The dysæsthesias of the mucous membranes are often limited to a very small area and must be searched for.

The *organs of special sense* appear themselves to be liable to a dysæsthesia which may provoke spasmodic attacks. It is not uncommon to see hysterical subjects who have attacks following the stimulus afforded by a strong odor; in others the paroxysms are provoked by exposure to bright sunlight or by a loud reverberation. Gradenigo insists upon the frequency of otalgia, sometimes reflex, sometimes associated with auricular lesions, and occasionally accompanied by hemorrhages; and he believes in the existence of auricular hysterogenic zones.

The *viscera* which may be the seat of hysterical dysæsthesia are the organs concerned in generation. Ovarian dysæsthesia is well known from the descriptions of Piorry, Schutzenberger, Négrier, and Romberg. Briquet mistakenly regarded pain in the ovarian region as a myodynia of the abdominal muscles to which he gave the name of cœlialgia and which he regarded as frequent. The ovarian region is often the seat of spontaneous pain, of a sensation of weight, of heat, or of neuralgic radiations. These pains are increased upon the approach of the menstrual periods, especially during walking. Recrudescences of the ovarian pain are also among the precursory symptoms of an attack; the ovary becomes the seat of lancinating pains and seems to be the point of departure of the globus hystericus. Sometimes the pain is so severe as to prevent or seriously interfere

with walking, the patients are doubled up and make pressure on the abdomen in order to quiet the pain. Sometimes a deep pain is accompanied by dysæsthesia of a small area of the skin corresponding more or less exactly to the ovary; but this superficial pain is more frequently evanescent, being most marked at the approach of the paroxysms. By burying the fingers, united in the form of a cone, at the level of the anterior superior spine of the ileum as if we were trying to compress the internal iliac artery, we may reach the point where the pain is most intense. Sometimes by this procedure we may be able to feel the ovary, and in any case we excite characteristic phenomena of pressure on hysterogenic or hysterofrenatric zones, that is to say, that according to circumstances or to the intensity of the pressure we may excite the aura or arrest the attack. In general a light pressure causes an ascent of the globus, while progressively increased pressure arrests the attack. The pain which is called ovarian is in fact seated in the ovary, at least in some cases; I have noticed several times in hysterical pregnant women the ascent of the painful point during pregnancy and its rapid descent after delivery. In these cases, furthermore, we may feel an increase in volume of the ovary, corresponding to the painful point. M. Baraduc has shown me a patient in whom on vaginal examination a small movable tumor could be felt alongside of the uterus, and compression of this tumor provoked all the phenomena ordinarily following pressure in the ovarian region.

The *mamma* may also be the seat of a deep-seated dysæsthesia and play the rôle of a hysterogenic zone, but more frequently we observe cutaneous or subcutaneous zones around the gland; the lateromammary hysterogenic zones are perhaps the most frequent. The mammary dysæsthesia may be accompanied by a swelling of the tissues constituting the gland and surrounding it, and by a redness of the skin.

The testicle may be affected in the same way as the mamma and the ovary. Testicular dysæsthesia may be symmetrically located on both sides, but it is more commonly unilateral and is always more pronounced on the side of the most marked sensory and motor troubles. In a certain number of cases a painful testicle presents certain malformations, either in volume or in position or in its relations to the epididymis. Very commonly the spermatic cord is also painful and the sensation caused by pressure of the cord passes up into the pelvis so that the region which corresponds to the ovarian region in woman may even in man be found dysæsthetic. Neither section of the cord nor ablation of the testicle causes the pain to disappear; it is well known that the same fact is noted after removal of the ovaries.

A knowledge of the dysæsthetic zones, especially of the hystero-

genic zones, which are also sometimes hypnogenic, is not without its use in a diagnostic sense. In a case reported by Pitres their presence enabled him to recognize the hysterical nature of convulsions occurring in a pregnant woman who was supposed to be suffering from eclampsia and upon whom it was proposed to induce an abortion. The discovery of the existence of hysterogenic zones may not only serve to reveal a latent hysteria, but may also enable us to provoke a discharge which is followed by a radical and sometimes happy change in the progress of the malady.

The dysæsthetic and hysterogenic zones may disappear under the influence of physical or mental stimuli. Pitres succeeded in causing their disappearance by inducing local ischæmia by means of an Esmarch bandage.

Paresis.—Various disturbances of motility correspond to those of sensibility. The greater number of hysterical patients have a certain weakness of voluntary movements to which the name amyosthenia is usually, but improperly, applied. This loss of power of hysterical subjects was well recognized by Briquet, and has been studied by Burq and Richer. It is ordinarily most marked on one side, usually the left, but it may be more limited; nevertheless, when it appears to be limited, it is very often generalized. This paretic condition coincides with various conditions of the tendon reflexes, which may be at least greatly weakened if not abolished, or normal, or exaggerated. This loss of power has generally a regional correspondence with the anæsthesia, which is also more common on the left side. These troubles of motility are influenced by a great number of physical and moral agents; the paresis is genera ly most marked at the time of waking, and it is increased in the dark. Under the influence of depressing moral emotions it may become exaggerated even to complete paralysis. I have elsewhere remarked ' that the night paralysis, described by Weir Mitchell, may be hysterical in nature. The loss of power of hysterical subjects is characterized not only by diminution of dynamometric pressure, generally most marked on one side, but by the form of the dynamographic curves which reproduce the curves of fatigue. This form is often found on both sides even when the paresis might be thought to be unilateral; and when we cause a transfer by the application of æsthesiogenic agents on the supposed healthy side, we often note that the first effect is an increase of power on this side, the curve of which reassumes the normal form. This fact shows that the paresis is general and that the æsthesiogenic agent has a general tonic effect.'[8]

Besides these troubles of motility we may mention hypotonia, or diminution of muscular tonicity, manifested by one-sided flaccidity

of the face. This flaccidity of the muscles favors relaxation of the joints and sometimes sprains, especially of the ankle.

Contractures.—Brodie remarked upon the facility with which contractures are produced in hysterical subjects as a result of very slight irritations, and Duchenne, of Boulogne, noted the existence of a tendency to temporary contractions from strong emotions in the hysterical; but it was Charcot and Richer [18] especially who called particular attention to this predisposition to contracture, this diathesis of contracture, which is seen in hysterics, recalling the reflex hyperexcitability associated with organic lesions of the pyramidal tract. This state, says Richer, resembles paralysis in that it coincides most frequently with a muscular weakness; it resembles contracture in that the slightest cause usually suffices to make it appear. In proportion as it is exercised this property increases; and although it has generally to be sought for, it may appear spontaneously at the moment of making a violent movement. Almost always, on the side of the contracture, says Charcot, there is a more or less marked anæsthesia, an ovarian pain, a certain degree of paresis, all relatively benign accidents, but which, as everything leads us to believe, have preceded the appearance of the contracture. The tendency to contracture, which is often exaggerated by hypnotism, may be brought to light by a great number of direct or indirect excitations; and it is not necessary that these be strong, for slight irritations of the skin, even on the more anæsthetic side, may excite contractions. Psychical irritations may also produce them. The most effective means of causing these contractures are, according to Richer: deep massage, repeated taps on the tendons, compression of a nerve, the application of a vibrating tuning-fork, faradization of the muscles or nerves, the application of a magnet, or simple touching of the skin. This last-named procedure enabled Richer to establish a somnambulic form which he opposes to a lethargic form obtained by the other methods. A strong voluntary contraction of the muscles may also provoke a contracture. The same effect may be obtained by circular compression of a member by a bandage, provided that the constriction is strong enough to irritate the muscles without going so far as to produce local anæmia, for then the contracture ceases. When the band which has provoked the contracture is removed, the latter may persist, disappear, or become generalized either in the muscles of the limb, in those of the whole side of the body, or even all over the body. The generalization occurs with especial ease in the regions already affected with anæsthesia or paresis; and the extension of the anæsthesia is always accompanied by extension of the contracture. Richer has noted that in the contractured members there is always an exaggeration of the tendon re-

flexes, and sometimes an epileptoid tremor. The affected muscles have preserved their electrical excitability, but the effects of electrical stimulation undergo interesting modifications; we notice an extension of the electrical stimulation under the influence of rapid excitations, and an increase of shocks produced by slow excitations; and in addition there is a sort of abnormal contraction of the muscle characterized by a prolongation of the descent which sometimes may even be incomplete so that the electrical contraction is followed by a permanent contracture. Under the influence of repeated electrical stimuli, this contracture may invade the entire member. The condition of contracture does not involve a maximum shortening of the muscle, and the limb may retain the position it had before the contracture appeared.

The liability to contracture is not limited to the voluntary muscles; and spasms of the œsophagus and of the neck of the bladder may be regarded as manifestations of this same tendency to contracture.

Contracture is closely related to anæsthesia and paresis, which may precede the contracture and remain after it has disappeared. So long as the diminution in sensibility and motility has not disappeared, so long has the tendency to contracture not disappeared; this tendency persists, altough undergoing variations in intensity, and this explains the frequency of a return in the contractures.

Contractures, especially recent ones, are often happily influenced by massage or moderate compression, or by the application of the magnet, which may cause them to disappear rapidly. Contractures of long standing are very obstinate and may resist treatment for years, hence the wisdom of Charcot's advice not to let contractures drag along. We must not neglect those which have appeared after hysterical attacks any more than those which have been provoked by other causes.

The *eye* is the organ in which are best shown the relations which exist between the troubles of sensibility and those of motility. We have seen that the sensibility of the eye in all its forms is affected in hysteria, as is also the sensibility of the teguments of this organ. The sensation of movement is also affected; Borel[24] has shown that, if we try to make the eye of an hysterical subject follow the movements of the axis of a cylinder, the patient has no idea of the direction in which his eye looks; the loss of the muscular sense may then be as complete for the muscles of the eyes as for those of the extremities. This loss cannot be without influence upon the functions of the ciliary muscle. According to Parinaud, Brücke's muscle (the longitudinal portion of the ciliary) is the one most frequently affected in hysteria, its contracture is always found when that of the orbicularis exists, but it may also occur alone. The contracture of the muscles of accom-

modation produces a sort of myopia which is marked by the circumstance that, because of the fixity of attitude, the eye can see objects clearly at one distance only. This contracture plays an important rôle in the production of monocular polyopia, of micropsia, and of macropsia; it is seen especially in hysterical subjects who are predisposed to contractures of other muscles of the body; but it also may present itself as the predominating if not the only contracture. It is this contracture which plays the principal rôle in the myopia of nervous children, and in the troubles of accommodation associated with certain diseases and with traumatism.

Among the contractures of the extrinsic muscles of the eye *blepharospasm* is perhaps the most frequent. This may be provoked by a slight irritation of the conjunctiva, such as that resulting from the presence of a foreign body, a mild inflammation, etc.; and like all the other spasms, it may follow a convulsive attack. It may also come on apparently spontaneously. Pressure on certain points, perhaps at a distance, may increase or arrest it. Blepharospasm may be present under a clonic or a tonic form, or as a pseudoparalytic ptosis (Parinaud). The clonic form is the most common; it is shown by a continual winking which is usually bilateral; it may occur in attacks or be a permanent condition; it may constitute the chief element of the morbid condition or it may coincide with other troubles. We sometimes see it during the attacks of sleeping, and according to Gilles de la Tourette it is a distinctive characteristic of these attacks and of hypnotic lethargy.

Tonic blepharospasm may be painful or not. When painful it is usually bilateral, although more marked on one side; it is often accompanied by photophobia and lacrymation; it is often very difficult to overcome and the more so as it often coincides with hyperæsthesia of the periorbital region and the eyelids. Painless blepharospasm is frequently unilateral; the forced closing of the eyelids is less energetic and is not accompanied by either lacrymation or photophobia. These two forms of tonic blepharospasm differ rather in intensity than in character.

Blepharospasm is usually accompanied by a depression of the eyebrow on the same side. This characteristic is wanting in pseudoparalytic ptosis of Parinaud, which consists in a simple falling of the upper lid without any sign of a forced contraction. This ptosis has really nothing to do with spasm and belongs rather to the ptosis of the nocturnal paralysis of Weir Mitchell, in which it is necessary to throw the head backwards in order to be able to see through the palpebral slit. This ptosis often coincides with other pareses of the same side. Finally we may note the fact that it is not always easy to

distinguish between the effects of contracture of the orbicularis muscle and those of paralysis of the levator palpebræ.

When the blepharospasm is very marked it is often accompanied by spasms of the motor muscles of the eye, and diplopia may result from it. It is also accompanied by sensory troubles of the eye, including all forms of visual anæsthesia, and also by disturbances of cutaneous sensibility under the form of systematic anæsthetic and dysæsthetic zones. In quite a number of cases the blepharospasm disappears under the influence of a more or less direct psychical treatment; and as a rule also it is to the general condition that we must address our therapeutic measures rather than to the local trouble.

We have seen that the blepharospasm is sometimes associated with spasms of the motor muscles of the eye, but the latter may also, though rarely, be alone affected. Hysterical strabismus has long been recognized, but it is only of late that it has been observed as a regular symptom. It usually occurs in the form of convergent strabismus, although all the muscles appear to be affected. We may also observe spasm of the associated muscles, and a spasmodic conjugate deviation has been noted.

It is not always easy to distinguish ocular paralyses from spasms, and indeed paralysis of the upper lid may exist at the same time with facial paralysis in hysterical hemiplegia. Hysterical ptosis as an isolated symptom has been seen but rarely, and its existence is doubtful. A paralytic conjugate deviation has, however, been observed. In a case reported by Parinaud there was coincidently with a paralytic ptosis a paralysis of accommodation with excessive dilatation of the pupil and a partial paralysis of the internal and inferior recti; but the paralysis of the motor oculi muscle would appear to be very rare in hysteria. Mydriasis and myosis seldom occur independent of other symptoms, but Giraud-Toulon has reported a case of spasmodic mydriasis. Paralysis and spasm of the sixth pair would appear to be exceedingly rare (Borel). The same is true of total paralysis, of ophthalmoplegia characterized by complete immobility of the eye. In a case reported by Bristowe there was double ptosis with almost complete immobility of the eyeballs, without mydriasis. This absence of mydriasis is the rule in hysterical ophthalmoplegia, which furthermore remains external and is never internal or total.

Tremor.

Paralysis and contracture are, however, not the only troubles of motility observed in hysteria. Ch. Lepois recognized tremor as a precursor of paralysis; and the history of miraculous cures comprises some cases of tremor which he regarded as really minute convulsions.

But even if we admit that there are attacks of tremor which may be regarded, like the attacks of contracture, as paroxysms, nevertheless we hold that tremor manifests itself more frequently as a permanent symptom; it would appear to be associated with a depressed condition of motor energy.

Hysterical tremor may be limited to one side of the body, present itself in a paraplegic form, or be generalized. Its characteristics are rather varied (Charcot, Pitres, Rendu, Dutil). It is rather infrequent as compared with other motor troubles, and is, according to Charcot, rather more common in men than in women; it may also be observed in children. As a rule, it appears suddenly as a result of some physical or moral shock, but still more frequently (Dutil) it follows a convulsive attack. The tremor, like the paralysis or contracture, is subject to periods of exaggeration and remission. Its duration varies greatly; it may persist for months without intermission, or its course may be very ephemeral. It is no less variable as regards intensity; sometimes hardly appreciable, it may in other cases be of sufficient intensity to impede the various functions. The rapidity of the oscillations also varies considerably; sometimes they are remarkably slow, at other times very rapid. Occasionally the tremor manifests itself only upon the inception of voluntary movements. Most commonly it persists during repose, but is more or less increased in frequency and amplitude by active movements and by various external conditions. The tremor not only varies in different individuals, but may also be variable in the same individual; it is what Dutil calls a polymorphous tremor. The slow non-intentional tremor, with four or five oscillations per second, resembles the tremor of paralysis agitans (Oppenheim, Ewart) or senile tremor; the rapid non-intentional form recalls the tremor of exophthalmic goitre, of alcoholism, or of general paralysis. Intentional tremor mimics that of multiple sclerosis and mercurial tremor, which latter Letulle regards as being often of an hysterical nature.

Vibratory tremor (Pitres) with rapid oscillations, eight or nine per second, is often a markedly convulsive and ephemeral phenomenon, but it may become a chronic condition. The patient is agitated by a perpetual tremulation; the muscles of the extremities and of the trunk are animated with fibrillary contractions; the face and the tongue may participate in the tremor when it is very intense, and the fingers are subject to sudden twitchings. This tremor ceases only during sleep. The disturbances of enunciation, the troublesome movements of the lips, and the trembling of the hands recall the features of general paralysis. When the tremor is very pronounced in the lower extremities it may simulate epileptoid trepidation, especially when the reflexes

are exaggerated. Following attacks this tremor may become exaggerated and may be accompanied by a loss of power in the lower extremities (Dutil). This weakness of the lower limbs may be seen in Graves' disease (Charcot), in the lumbar crises of locomotor ataxia (Pitres), and in general paralysis (Féré); such association may add to the difficulty of diagnosis, but it shows very clearly the paralytic nature of the tremor.

The most frequent tremors are those of moderate rhythm (Dutil), intermediate, as regards the number of oscillations, between the vibratory and the slow tremors. They vary in their extent and in the modifications which they undergo during repose. We may distinguish among them an intentional remittent type (Rendu, Pitres), resembling mercurial tremor and that of multiple sclerosis; with this difference, however, that instead of becoming manifest only upon the inception of voluntary movements it is permanent, but is exaggerated in intensity when the patient attempts to execute any movement whatever. It is a tremor generalized throughout the extremities, the trunk, and the head, even the tongue. The fingers, however, are as a rule affected only by transmitted movements and have no tremor in themselves; the other segments of the limbs are agitated by alternating movements of flexion and extension, which become exaggerated when the patient begins to walk or to execute any voluntary movement whatever. Hysterical intentional tremor has such a close analogy with mercurial tremor that Letulle was forced to the belief that mercurial poisoning would provoke tremor only in a predisposed individual.

Intentional tremor may be manifested chiefly on one side only, or it may be strictly hemiplegic; sometimes, however, it assumes the paraplegic form. When it is confined to the lower extremities it simulates the trepidation of spasmodic paraplegia; but the patellar tendon reflex is not exaggerated, and sometimes it is even weakened, and a sudden forced flexion of the foot will arrest the tremor instead of reinforcing it, as in spasmodic paraplegia (Dutil).

Alongside of this remittent intentional tremor there is a pure intentional tremor, of the same moderate rhythm, which is manifested only on the occasion of voluntary movement (Pitres), and which resembles very completely the trembling of multiple sclerosis. The speech, instead of being scanning, has the characters of hysterical stammering. But the study of the associated symptoms should be made with care, for hysteria may simulate many symptoms of sclerose en plaques, and besides the two affections may coexist. The variability of the hysterical symptoms may be of great assistance in the diagnosis, but the existence of this affection has passed unrecognized, under such circumstances, by some of the best-qualified clinicians

(Westphal, Langer, Francotte). Buzzard does not admit the existence of intentional tremor in hysteria, but thinks that the opposite is the fact, and that cases of multiple sclerosis have often been mistaken for hysteria. The influence of physical and æsthesiogenic agents may furnish us with information of great value in the diagnosis of hysteria. The attacks of tremor which I have studied in epilepsy may, in the absence of other diagnostic facts, lead to confusion.

Hysterical patients have also occasionally other forms of twitching resembling those seen in epilepsy; among them especially nystagmus, an affection which is common to the two conditions.

General Nutrition.

It has been remarked that very many hysterical patients have a poor appetite and sleep badly, but are, nevertheless, quite active and do not emaciate. It might be premised that the functions of digestion, secretion, circulation, and respiration would not be performed as in normal condition. Certain cases of hysterical anuria in which urea has been found in the vomited matters are instances in point. Empereur,[21] in a study of hysterical subjects suffering from digestive troubles, concludes from an examination of the excreta that, in general, in hysteria the nutritive processes are retarded and that assimilation does not occur because disassimilation does not take place. He admits, however, that in certain hysterics who present no morbid symptoms nor permanent stigmata there are no disorders of nutrition. Gilles de la Tourette and Cathelineau conclude as the result of their examination of the urine that in hysterical subjects, apart from pathological manifestations of the neuroses other than the permanent stigmata, nutrition is effected normally, the volume of the urine and the amount of urinary excreta in proportion to the weight of the individual being exactly the same as in healthy individuals.[22]

According to Briquet, of all the older writers who studied the condition of the blood in hysterical subjects Ettmuller is the only one who held that it was thick, all the others stating that there is a diminution in the solid elements. Quinquaud concluded as a result of his investigations that there were no changes in the blood in hysteria except in those who were chlorotic.[23] Gilles de la Tourette and Cathelineau[24] found that cutaneous incision in a hysterical subject would give issue to an amount of blood one-third less than a similar incision would give issue to in a healthy subject, and that apart from anæmia and chlorosis the quantity of hæmoglobin, of urea, and of glucose is normal.

Hénocque has devised a procedure for studying the time necessary for the reduction of oxyhæmoglobin in the tissues. I have observed

that in hysterical subjects the time of reduction is longer on the anæsthetic side. This reduction time undergoes important variations under the influence of peripheral stimulations or of emotions. The researches which I have made also reveal a small amount of oxyhæmoglobin in the blood. This is a condition which is common to most neuroses."

Mental States.

An error which has long offered an impediment to a comprehension of the mental state of hysterical subjects lies in the belief that simulation and deceit fill all their psychical life. Charcot, Pitres, and Gilles de la Tourette have justly combated this opinion. In reality hysterical subjects express false judgments because they perceive incorrectly and consequently reason wrongly. A study of the general and special sensibility in these patients explains well the genesis of their errors. In fact, it is the man who is the measure of all, and hysteria is a measure which is not only false but essentially variable and under the influence of surrounding conditions and even of conditions created by the sensations. The disorders of sensibility are quite capable of giving rise to errors of judgment concerning the actual surroundings. But it is not only false perceptions during the waking hours which disturb the ideation; the recollection and the associations of dreams may still further complicate the situation. The sleep, often insufficient, of hysterical subjects, is ordinarily troubled by dreams of a nature to provoke sadness or fright. Now it often happens that these dreams remain in the memory during the waking hours and fix themselves in the imagination with such an intensity that the patient is incapable of distinguishing the reality. Furthermore we know that these representations cannot exist without the coincidence of somatic conditions which necessarily accompany the actual sensation; that is to say, that corresponding to these very intense representations there are characteristic somatic states which may consist in anæsthesias, dysæsthesias, paralyses, contractures, or disorders of nutrition of the most varied kind. Such is the pathogenesis of the suggestive phenomena which may arise either from external stimuli or from dreams or waking delusions. There is no question that the rôle of the imagination is considerable in the pathogenesis of traumatic hysteria; but to hold that the imagination is the only active factor is to exceed the limits of probability, for we cannot hold that shock itself is powerless to do what may be done by the mere representation of shock. In many cases the imagination merely accentuates the effects of the shock, it completes them. I do not mean by this to deny the effects of imagination, for I have myself seen a para-

plegia consecutive to a dream, and we are often able to observe the influence of a dream upon delirium in cases of hysteria.

What is done by the dream may be reproduced by the post-paroxysmal delirium, by an external irritation, or even by a simple suggestion. Dreams, especially erotic dreams, which may be accompanied by physical phenomena, play an important rôle in the false accusations brought by hysterical patients. Hysterical subjects have, as a rule, an extraordinary credulity, which accords well with their loss of sensibility and the weakening of their powers of discernment; they can with difficulty establish the difference between actual sensations, the representations of a dream, and the suggestions to which they have become subjected.[26]

The reflex irritability of hysterical subjects increases in proportion as the power of control, the will, diminishes; they are subject to irresistible impulses which have the peculiarity of being easily exhausted. It is this which has led observers to attribute the quality of simulation to many of their acts, for example, to the attempts at suicide which, however, succeed occasionally. I have seen an hysterical woman throw herself from the window of the third floor of the Salpêtrière, breaking both thighs; the motive for the act was very slight, but the woman could have had no illusion concerning the consequences of her act.

Although it is true that the false assertions of hysterical subjects do not always constitute actual lies, but rather are the result of the imperfection of their sensations and of the deductions which they draw from them, nevertheless they do sometimes lie roundly. Under the influence of the disease images become more intense to them and those created by autosuggestion present themselves to their minds as realities; it is only when a period of calm has returned that they can see that there is nothing true in what they have just before imperturbably maintained. Painful hysteria often gives rise to manifestations suggesting simulation; but commonly enough we can recognize physical phenomena of such a nature as to put us on our guard against an arbitrary scepticism.

It is the weakening of the nervous functions which causes the troubles of motility and of sensation in hysterical subjects; it is their irritability which renders them insupportable and serves as a base for their impulses, and their suggestibility which gives them the appearance of professional liars; it is also this weakness which gives to their character its general tone of sadness. This sadness, which is more marked in men, a great number of whom have made or premeditated attempts at suicide, is increased under the influence of all the physical and moral conditions which are capable of causing a depression of the

nervous functions. Mental action is a function of connection; it would appear that in hysterical subjects the connections of the neurons are relaxed.

Reaction Time.—The disorders of sensation and of movement so frequent in hysterical subjects are of a nature to lead to the assumption that there are also important modifications in the reaction time. However, this fact had not been definitely established at the time when I began the study of the subject some years ago. The duration of simple reaction time in hysterical subjects is usually increased in proportion to the diminution of general and special sensation; it varies for each sense. The reaction to cutaneous stimuli may be more or less retarded according to the region of the body. The duration of the reaction time is shorter when the stimulus and the reaction are on the less anæsthetic side; it is increased when the stimulus only is made on the more anæsthetic side or when the reaction occurs on this side; it is increased also when the stimulus and the reaction are both limited to the more anæsthetic side; that is to say, the centripetal and centrifugal transmissions are more retarded on the more anæsthetic side. In hysterical subjects the effect of fatigue upon the reaction time is much more marked than in normal individuals; the time varies also very considerably under the influence of sensory stimuli or emotions. In hysterical subjects the association time is retarded, as I have noted, very considerably, and this retardation varies under the same conditions and in the same sense, but not in the same proportion as the energy of voluntary movements, sensations under all its forms, and the reaction time. Exercise, fatigue, physical excitation, and emotions have a marked influence upon all forms of activity in hysterical subjects.

Paroxysmal Phenomena.

The greater number of the persisting or permanent phenomena which we have just passed in review may remain unperceived; those which remain to be studied are very evident; these are the hysterical symptoms which manifest themselves under the form of paroxysms. But these paroxysms must not be understood as necessarily rapid manifestations, for they are on the contrary often quite prolonged. Their common characteristic is that they may arise suddenly and disappear in the same way, whether their duration has been a few minutes or several years.

Convulsive Attacks.

The most common of these hysterical symptoms are of the convulsive order. Hysterical convulsions may occur at all ages; they are

observed in nearly three-fourths of the female patients (Briquet), but in the case of men these proportions are reversed (Pitres). Although the convulsive symptoms are much more rare in patients of the male sex, they are nevertheless often much more grave in them. In children the convulsive attacks are ordinarily abortive, but they become more complete as age advances. The convulsive rages of children have been attributed to hysteria (Chaumier), but they are at least as commonly preludes of epilepsy and we are the less able to make a differential diagnosis for the reason that hysterical stigmata are, as a rule, wanting in childhood. The tendency to convulsive attacks grows less after the age of forty years, but although the attacks then become more rare, we cannot say that they are absolutely wanting in old age;[27] they may be observed in the aged of both sexes.

It is somewhat difficult to decide whether these attacks may constitute the first manifestation of hysteria, for there is always doubt concerning the preëxistence of stigmata. When the attack is the first symptom observed it is usually manifested as the result of some moral or physical shock; sometimes this shock may be caused by a dream.[5] Acute intoxications, especially acute alcoholism, may play the same rôle; and the induced sleep of ether, chloroform, or hypnotism may have the same effect.

In hysterical subjects already showing stigmata, the first attack may be provoked by the most trivial causes; nevertheless it often appears under circumstances of a general weakening, as by acute disease, fatigue, or worry. Whatever may be the conditions under which the first attack is produced when once the system is primed, the explosion may take place under the influence of the slightest cause; hence the rule to prescribe quiet and isolation. Those patients who have attacks less frequently often have them more severely, as we see in the case of men, yet this is not a general rule. That which is more constant is that in the same subject the attacks are stereotyped, presenting themselves always under the same form. The attacks of hysterical convulsions are almost always diurnal; even when they are provoked by a dream the discharge generally takes place in the morning. Sometimes the attacks recur constantly at the same hour, especially when we have to do with collective manifestations or those which take place under the influence of some external circumstance that is regularly repeated. The menstrual epoch is favorable to the production of attacks, chiefly, however, when the uterine functions are disordered.

Prodromes.—The convulsion may have a sudden apoplectiform onset with loss of consciousness, but more commonly it is preceded by premonitory trouble often constituting a true aura and repeated, as a

rule, in the same subject with a remarkable uniformity. The premonitory signs of an attack are mediate or immediate, they indicate a danger which is more or less distant or one which is urgent. The first are modifications of character, the patients becoming irritable, presenting alternations of excitement and depression, approaching, especially in man, to a condition of apathetic moroseness. This state, which is often accompanied by perversions of taste and of appetite, may last for a few hours or several days, and while it lasts sleep is generally disturbed and dreams are troublesome. During this period the patients may in a certain measure struggle against the attacks and retard its appearance. Just as in epilepsy, moreover, this prodromic period may stop short and the attack does not then appear. There is hardly any opportunity for a struggle on the part of a patient against the immediate premonitory phenomena; his will is powerless and resistance, moreover, is distressing; the patient prefers to have a convulsion rather than to fight against it, and he looks for the most convenient and least dangerous place in which to have it.

The most frequent of the immediate premonitory phenomena is without question an exaggeration of the ovarian or testicular pain; but in general all the dysæsthetic or hysterogenic zones show an increase in their morbid sensibility. Whatever may be the location of the zone the sensibility of which is exaggerated, provided that it is subepigastric, a globus starts from it, mounting towards the pit of the stomach and then to the throat. This sensation is accompanied by a feeling of oppression, of constriction of the pharynx; then pulsations appear in the temples, ringing and whistling in the ears, especially on the side most affected by the stigmata, and the attack breaks out. Sensations of suffocation and of constriction of the throat which characterize the aura appear to be accompanied by true spasms of the muscles; deglutition is often impossible. Sometimes this sensation of the hysterical globus is accompanied by vasomotor phenomena, such as redness of the face, local anæmia of the extremities, with a sensation of formication, and a paretic state.

For a long time the hysterical convulsive attack was regarded as an essentially variable trouble in some sort impossible to describe. Charcot has brought a little order out of this chaos by showing that the various phenomena of the hysterical attack do not defy classification, and that all the varieties of convulsions might be brought together under one type of which he has given a classical description; but the subject has also been profitably studied by several of his pupils, Bourneville and Regnard, and especially by Paul Richer.

Epileptoid Stage.—Whatever have been the prodromes, the attack

of hystero-epilepsy (grande hystérie of Charcot) commences with an epileptoid period. The patient sinks down or falls over, without crying out, but sometimes making an audible hoarse noise in the pharynx, produced by a sudden inspiration. The loss of consciousness is complete, sensation is abolished even though it may have been exalted during the prodromic period. Of this phase there is no recollection. The tonic convulsions which begin immediately may be general, but they are most marked usually on the side on which the stigmata have predominated. The face is pale, the neck stiffens, the head is thrown back, the throat becomes swollen, the shoulders are elevated, and there is congestion of the head. The eyebrows are elevated, the eyes are convulsed, looking obliquely upwards, the mouth is opened, and the tongue is often projected forwards or laterally; the teeth are rarely closed, and it is altogether exceptional for the tongue to be bitten. The glottis closes after inspiration and the chest and abdomen remain swollen, the neck is also tumefied, and from time to time there are brusk respiratory movements. The swelling of the neck coincides with a congestion of the surface and a turgescence of the subcutaneous veins, which may be seen standing out under the skin. All these phenomena are necessarily transitory, as are also the respiratory spasms which give rise to them.

The upper extremities execute slow movements of circumduction; the fingers are flexed over the thumb, the wrists are flexed in pronation, the arms are raised, and the elbows are flexed, bringing the hands before the face; then the members are extended again along the body, the fists being in forced pronation. The trunk becomes twisted, and is alternately flexed and extended. The lower extremities also execute alternate movements of flexion, extension, and rotation. These movements, which are as a rule very slow, last but a short time and are followed by a general tetanic spasm. The extension of the head becomes exaggerated, the swelling of the neck increases still more, and the face is puffed out and cyanosed. The upper extremities are extended in adduction and rotation outwards, the wrist being flexed, the fist tightly closed. The lower extremities are extended, and the feet are in the position of equinus, turned inwards or outwards. The trunk is fixed, often in opisthotonos; sometimes the body, strongly curved backwards, rest only on the heels and the sinciput, forming the arc of a circle. This description does not correspond strictly to the facts in every case; sometimes, during the epileptoid period of the attack, the members may assume the position of crucifixion.

To the tonic contractions soon succeed clonic convulsions, which as a rule begin in the member which was the first tetanized. The

face makes grimaces, the head and the extremities become animated with rapid oscillations, the respiration is resumed, but is whistling and staccato; the abdomen has independent and irregular movements of its own, and sometimes borborygmi are audible.

To this clonic stage, to which we are unable to assign any definite duration, succeeds a stage of muscular resolution, characterized by complete relaxation of all the muscles; the patient lies sunken down in the dorsal decubitus, the head lies on one shoulder, the face is swollen, the eyelids are trembling, and the respiration is stertorous. Sometimes resolution is incomplete and contracture occurs in a limb or in the entire half of the body, but as a rule this contracture disappears during sleep.

The duration of the epileptoid period is, on an average, according to Richer, about sixty seconds for the two tonic and clonic stages, but there are very important variations from this. The length of the period may be increased or modified by the absence of one of the stages. I have several times observed before the onset of the convulsion, without any trace of a tonic contraction, a very active pupillary contraction constituting a sort of masked epileptoid stage, showing that the attacks may be more incomplete in appearance than in reality. The condition of the pupil furnishes an interesting subject for study.[12] During the attack the pupil is but slightly influenced by the action of light, which sometimes, however, induces a very marked contraction. When the face begins to be set by the tonic contraction preceding the torsion of the head, we see the pupil contract rapidly and then remain immobile. During all the first part of this first phase, during the tonic period, the pupil remains contracted. As soon as the clonic movements begin the pupil becomes strongly dilated, and this dilatation persists during the second part of the epileptoid period and all through the stage of strong convulsions.

Stage of Clownism.—The epileptoid stage is followed by stertor of usually short duration, then the attack arrives at the period of clownism (Charcot), of which we may distinguish two stages, that of illogical attitudes and that of extensive movements.

The illogical attitudes present the most bizarre characters, but there are several which occur with considerable frequency and have received a special decription, such, for example, as the "arc of a circle," which may be anterior, posterior, or lateral. Opisthotonos or the anterior arc of a circle is the most common; the patient rests on his head and heels, the upper extremities being extended alongside of the body, or else thrown up each side of the head. The abdomen projects strongly upwards, often having movements resembling those of cynic spasm, and which the older writers wrongly interpreted

as indicating sexual excitement. Sometimes the head is so far thrown back that the face rests on the ground; sometimes the feet are so sharply extended that the toes rest upon the ground. In order to form the posterior arc the back rests upon the ground, the lower extremities are raised and the trunk is curved in the position of emprosthotonos. At other times the curve is a pleurosthotonos—it is a lateral arch. These attitudes are maintained for a varying length of time, and with such force that the observer may rest his entire weight on the patient's body or limbs without moving them at all unless he happen to press on a hysterogenic zone. The arc of the circle may be maintained for ten or fifteen minutes, but more frequently it is a transitory attitude. It is sometimes accompanied by partial contractures; as a rule the jaws are closed and the face remains fixed.

When the muscles become relaxed and the body has sunk back to the position of dorsal decubitus, we observe the beginning of the stage of extensive movements which are characterized by their variety and extreme violence; the patients cry out vociferously, and the movements in general appear to be to enable the patient to escape or to defend himself from urgent danger.

Stage of Emotional Attitudes.—The third stage of the attack, or the stage of emotional attitudes, is the logical sequence of the preceding. The urgency of the symptoms has now ceased, although the representations in the patient's mind still remain very vivid; the voice and the gesture indicate anger or the greatest gayety or, more commonly, sadness. We may recognize the existence of hallucinations by the attitudes which the patient assumes, as we see him listening attentively or seizing imaginary objects. Sometimes we see very remarkable oscillations of the pupil, alternations of dilatation and contraction which vary with the attitude of the patient and seem due to the necessity of modifying the accommodation according to the varying distances of the objects or persons which figure in his imagination. The period of passional attitudes constitutes a sort of dream in action. This dream, which the patients do not always reveal, has generally a connection with the circumstances which have provoked the first hysterical paroxysm, such as a rape, or attempt at murder. The knowledge of the dream enables us to interpret easily the passionate attitudes which may express the most varied sentiments. Hallucinations of animals, zoopsia, often give to these attitudes a special character.

Stage of Delirium.—Following the stage of emotional attitudes comes the fourth period, that of delirium, which is in general characterized by a sad delirium which is based upon the representations of the preceding stage. The ordinary state of sensibility of the sub-

ject may have an influence on the representations of this delirium. Charcot has noted, for example, that according as the anæsthesia is located on the left or right side of the body, the animals which figure in the delusions pass from left to right or from right to left. Achromatopsia, if it exists, is shown in the hallucinations; red being the color the perception of which persists longest, it is the red which figures most frequently in the hallucinations. But external conditions are not without influence on the delirium. Mesnet has observed that contact with certain objects or variations in the light may increase it; this influence of external stimuli has been specially studied by G. Guinon and Woltke,[25] who conclude from their experiments that in the delirium of the passionate stage of an hysterical attack we may modify the course of the hallucinations and create new ones by means of diverse but always simple excitations of the organs of special sense, and that the hallucinations are invariably independent of the will of the operator and remain exclusively in the initiative of the patient, who appropriates the sensation perceived and transforms it as he will into an hallucination corresponding to his habits, his manner of living, his recollections, or, in a word, to his own personality. This influence of the surroundings is furthermore not peculiar to hysterical delirium, for we find it frequently in alcoholism.

The duration of the delirious stage is exceedingly variable; it may, like the other stages, be of relatively long duration, or may even occur independent of other manifestations.

Post-Convulsive Stage.—In coming out of the stage of delirium the hysterical patient does not return wholly to the normal condition. Total anæsthesia is the rule during the continuance of the attack, and sometimes this anæsthesia disappears, leaving only the troubles constituting the permanent stigmata, as soon as the delirium has ceased. More frequently, perhaps, there remain traces of the anæsthesia of the attack, and it is not rare to see a more or less complete and more or less lasting deafness or amblyopia remain after the convulsions have ceased. At other times we see remaining a contracture or a paralysis to which are generally added disorders of sensibility. A short time after the attack has ceased we almost always see an abundant discharge of clear urine. Patients may suffer from more or less severe fatigue, but seldom from any mental dulness such as is so common after an attack of epilepsy. Sometimes they even experience a feeling of very manifest euphoria, having a sensation of relief; as a rule they can rise without assistance and resume their ordinary occupations.

While the epileptoid stage hardly exceeds two or three minutes, the others have a very variable duration; most commonly the attack

as a whole lasts for from twenty to thirty minutes; but frequently the attacks are repeated at short intervals, constituting a true morbid status. The attack often modifies very considerably, either for good or for ill, the patient's habitual state.

The hysterical attack, such as Charcot described it, has been observed many times outside of the Salpêtrière, and reports have been published, both in France and elsewhere, showing that this is not altogether a cultivated form of hysteria (hystérie de culture). However, we must recognize with Richer that it is impossible to deny the rôle of imitation; neither can we maintain that this hystero-epilepsy (grande hystérie) is the most common form of convulsive hysteria. From the point of view of its convulsive forms as well as from that of its permanent manifestations, hysteria cannot be regarded as a complex of symptoms one and indivisible; whatever may be the advantage of our modern schematizations, hysteria remains the Proteus of the ancients, and we have no right to refuse the epithet of hysterical to all convulsive manifestations which do not agree in their features with the drama in four acts and a prologue which we have just sketched. Furthermore, by admitting with Charcot himself that there are larval forms of infinite variability, we can, in a manner satisfactory to all, reconcile the idea of a protean hysteria with that of an hysteria one and indivisible.

Diagnosis.—The attack of hystero-epilepsy, when it is complete, is characteristic, but not when it is incomplete. The emotional phenomena of the onset, which may remain the only ones, may be confounded with the anxieties and the phobias of the neurasthenic and the degenerate. It is especially difficult to differentiate the spasms of the epileptoid stage from those of true epilepsy. In a typical case the diagnosis is easy; as when, for example, the attack comes on during the night or in the morning, when the fall is sudden and is accompanied by a cry, when the patient bites his tongue and urinates in his clothing, when he always loses consciousness, and when he remains stupid and greatly fatigued after an attack; but there is no one of these characteristic symptoms which may not be absent. We may base our diagnosis upon the course of the affection, and especially upon the existence of permanent stigmata; but hysterical stigmata may be wanting in great part, and epileptics also often present permanent disorders, especially of sensibility, which may lead us into error. Indeed, in some cases, we are forced to reserve our diagnosis, at least for a time.

The Urine.—Gilles de la Tourette and Cathelineau believed that they had discovered a pathognomonic characteristic in the urine. It has been said that hysterical polyuria is relative and not actual, that

is to say, that the total amount of urine passed in the twenty-four hours during which the attack has occurred is not increased, indeed it is rather diminished. The increased quantity is noted only during the first micturition following the attack, and it is only at this time also that the lowered specific gravity and the fainter odor and color are noticed. But the most interesting facts are those relating to chemical analysis. It is asserted that the convulsive attack reduces by a third on the average the weight of the fixed residue. The amount of urea is said to be diminished in the total quantity of urine passed, but especially in that voided immediately after the attack. The total amount of phosphoric acid is also reduced from 2.19 gm., the normal, to 1.24; and instead of the normal proportion of 25.04 per cent. of earthy acids, we find 80. The amount of earthy acids is, in the normal condition, to that of alkaline acids as one to three, but this proportion tends to become as one to one during the attack. The amount of chlorides and of sulphates is said also to be constantly reduced. These changes in the urine are found even when the alimentation has been in no wise modified on the day of the attack. These characteristics are of the greater value since Gilles de la Tourette and Cathelineau found them in the larval or abnormal forms of hysteria, and since they differ markedly from the condition found in cases of epilepsy; in the epileptic attack the urine shows an increase in the amount of the fixed residue, of urea and of phosphates, but the relative proportions of the two latter remained normal (Lépine and Jacquin, Mairet). The question, however, is not to know whether these modifications actually exist in the convulsive stages of hysteria, or whether they exist frequently at these times, but whether they are characteristic. It is certain that the diminution of the excreta and the inversion of the formula of the phosphates is encountered in the convulsive period of hysteria, but these changes are not constant and they do not belong exclusively to hysteria. What appeared to be the most characteristic in the formula of Gilles de la Tourette and Cathelineau was the inversion of the formula of the phosphates. Some observers were satisfied with these researches, but Voulgres has found the inversion of the formula of the phosphates in locomotor ataxia, phosphatic diabetes, and epileptic convulsions. I made a number of observations in my hospital service and found the same inversion in a case of epileptic apathy and in one of epileptic vertigo, and believe therefore that I am justified in saying that the inversion of the phosphates may be found in several forms of epilepsy.

In view of these facts, Gilles de la Tourette has stated that the inversion of the formula of the phosphates is not in itself characteristic of the convulsive period of hysteria, but that there is also an increase

in the chlorides. But this increase does not belong exclusively to hysteria, and may also be wanting. The diminution of urea is also not constant, as has been observed several times in my service (Royer). Gilles de la Tourette has objected to these last-mentioned observations that they were made on insane hysterics, but, as I have elsewhere remarked, he was misinformed as to this. Not only may the urinary formula, which we have just been considering, be found in other affections than hysteria, especially in epilepsy, but it is also subject to variations in the same hysterical subject. Poëls has found the inversion of the formula of the phosphates in hysterical subjects at other periods than during the attacks. Gilles de la Tourette admits that here as elsewhere we may meet with exceptions,[29] but he has made a new series of analyses in a large number of cases, and has been able to confirm the results obtained in the former series. However, to prove that the urinary formula in question exists most frequently is not to prove that it is a pathognomonic sign, the character of which is not alone to be frequent, but to be at once necessary and exclusive.

While we see, therefore, that a chemical analysis of the urine does not enable us to reach a certain diagnosis, we find no surer guide in the toxicity of this fluid. In the course of my researches upon the toxicity of the urine in epileptics, I noted that there was a very feeble toxicity of the urine in an hysterical subject during the paroxysmal period. Since then, Bosc has also observed a relatively weak toxicity. But apart from the fact that the experiments in this direction were too few to be of any value, the methods employed to determine the toxicity were not free from suspicion.

Irregular and Larval Forms.

The attacks of ordinary hysteria (petite hystérie) present a very great variety of forms, which some writers have sought to compare to abortive forms of hystero-epilepsy (grande hystérie), from which the convulsive attack has been in a manner dissociated. This may be useful as facilitating description.

The *prodromic period*, characterized by perversions of appetite or even complete anorexia, sadness, and the desire for solitude and silence, variable emotivity, etc., may exist alone and constitute in itself the entire attack. On the other hand, this period may be absent, the aura appearing suddenly as a sensation of oppression; or sometimes the attack is accompanied by suffocation, by pains simulating angina pectoris or asthma, by ringing or whistling in the ears, or by a feeling of dizziness resembling Ménière's vertigo (Gilles de la Tourette). At other times, again, the aura is represented by an in-

crease of the pains in one or several dysæsthetic zones, constituting neuralgic attacks of variable seat. These forms of painful hysteria are accompanied sometimes by vasomotor phenomena, well calculated to dissipate all idea of simulation.

The aura may be wanting, the loss of consciousness manifesting itself as the initial phenomenon. There are syncopal (Briquet) and vertiginous attacks, which so strongly resemble homologous attacks of epilepsy as to be mistaken for them; their true nature may be recognized by other signs and especially by the absence of the apathy coming on after the attack. I have observed a young girl affected with hysterical losses of consciousness repeated twenty times an hour without any injury to the intelligence.[30]

The *epileptoid stage* may be wanting in the attacks of hysteria minor, but sometimes it constitutes the entire attack. Often the spasm occurs alone without any loss of consciousness. The attacks of spasm, preceded by a condition of malaise with depression of spirits, consist in rigidity accompanied by immobilization of the chest with a sensation of anxiety and suffocation, and deglutition is generally impossible. When the sensation of suffocation arrives at its height, the limbs move convulsively. Then follow headache, sobs, and tears, the attack terminating with a feeling of fatigue which is succeeded usually by a sense of extreme well-being. This agreeable sensation of relief may be wanting, and the crises may recur in the form of a series or there may be a permanent condition of malaise. The crises of suffocating spasm may sometimes be accompanied by urgent danger; in a case of this sort, reported by Muselier, it was necessary to perform tracheotomy and to leave the cannula in position for two months. In the rare cases in which death occurs in consequence of the attack of hysteria, this spasm appears to hold a predominating position among the causes of the fatal issue.

The typical epileptoid stage of an attack of hystero-epilepsy may occur as the sole manifestation of hysteria minor, or sometimes there is a series of epileptoid spasms separated by variable intervals, and these may, like the attacks of hysteria major, recur daily for months. At other times these epileptoid attacks recur in rapid succession, without normal intervals, as a status hystericus (*état de mal*—Charcot, Bourneville), which may be distinguished from the status epilepticus, especially when the convulsions are limited to one side, only by the absence of fever. The absence of tongue-biting and of involuntary evacuations is not exclusively characteristic of the hysterical condition. The epileptoid hysterical crisis may assume the facial, brachial, or crural types, or that of partial epilepsy from cerebral lesion.

Sleeping Attacks.—In hysterical subjects we sometimes observe sudden but short-lived attacks of deep slumber, which Gélineau has designated by the name of narcolepsy, but which are always symptomatic, as Ballet has well shown.[31] These crises might be compared to the post-epileptoid stertor, and thus be regarded as a larval form of hystero-epilepsy. We may also include in the stage of muscular resolution and of stupor most of the soporific attacks which have attracted attention from the earliest times (Galen) and have been described variously under the names of lethargy, apoplexy, drowsiness, syncope, and apparent death. Sleeping attacks occupy an important place in demonomania. They have been regarded as apoplectiform or syncopal attacks, but of late have been interpreted as a larval form of the hystero-epileptic attack, in which the somnolent phenomena assume an exaggerated importance.

The sleeping attack is rare in the young, but may occur in both sexes in middle life and even in old age. The apoplectic form is more common in men. A sudden onset of the attack is rare in women, but apart from this it occurs, especially in the narcoleptic form, in them as well as in men. Most frequently the attack is preceded by prodromes analogous to those which announce the other attacks; but these prodromes are not of much assistance in the way of averting the threatened attack. Pitres has reported the case of a woman who had to yield to the sleep, even when she ran about to prevent it or put herself under the shower bath.

Certain syncopal or vertiginous attacks may be regarded, in some cases at least, as mild sleeping attacks; but more frequently these attacks are of very long duration, so that they may really be looked upon as constituting a sort of status hystericus (Gilles de la Tourette).

In whatever way the attack has come on, whether isolated or consecutive to other paroxysmal manifestation, the attitude of the patient is that of one in a profound and peaceful slumber without stertor. As the sleep is prolonged the face grows pale and the extremities become cold and may sometimes appear cyanotic, giving to the person almost the appearance of a corpse. When the pallor is replaced by a congestion of the face it is often the prelude to an interruption of the sleep by some intercurrent trouble. The muscular resolution is often incomplete and replaced by contracture, either limited to certain muscles, as the masseters, which are rarely unaffected, or generalized. This generalization of the contracture may persist for weeks without appearing to interfere with the functions of nutrition. Often the eyelids, instead of being drooping and without folds, as in normal slumber, are the seat of a spasmodic contracture which gives place to

a more or less continuous tremor. If we endeavor to overcome this contracture in order to ascertain the condition of the pupils (which is variable), we often excite a contracture of the motor muscles of the eye, if it has not existed previously, so that the eyes are deviated inwards and upwards.

Respiration is generally slow and superficial, sometimes it presents the Cheyne-Stokes type, or more rarely it is rapid at intervals. These momentary accelerations may be in relation with emotional states occurring during dreams. The pulse is usually calm and regular, rarely slow or rapid. The temperature remains normal. The general sensibility is usually abolished, and often the hysterogenic zones, if any exist, become unexcitable or at most provoke only a few spasmodic movements or some defensive motions. At the end of the sleeping attack the hysterogenic zones recover their activity, and irritation of them may provoke intercurrent convulsive crises. All the stimuli of the special senses may also remain without reaction. As a rule, after the attack, the patients retain no recollection of these various excitations, but exceptionally they remember what has occurred during the attack and obey its suggestions. The impressions received by the organ of hearing are those which are the most often retained. In a certain number of cases the sleep is so troubled by dreams that we may judge of their character from the changing expressions, by movements of defence or attack, etc. The sleeping attacks are often interrupted by spasmodic movements of various kinds. They are of variable duration, lasting from a few minutes to some hours, or even weeks or months; they may occur as a series in rapid succession, and relapses are frequent. Sometimes the patient emerges from the attack as quietly as he would wake from a normal slumber, but more commonly the attack terminates by a spontaneous or provoked convulsive crisis. Not uncommonly the hysterical slumber gives place to other convulsive or paralytic troubles. Hysterical sleep has given occasion to premature burial, but such is hardly excusable even in the condition of apparent death; there is no authenticated case of a fatal termination of an attack of this kind. Except in those cases in which the sleep is interrupted by momentary periods of more or less complete awakening, it will be necessary to resort to artificial alimentation, preferably by the œosphageal sound. This artificial feeding sometimes keeps the patients in good condition without emaciation for weeks, but as a rule there is a loss in weight at the same time with a diminution in the excretion of urine and of urea (Gilles de la Tourette). Sometimes the excretion of urine and the amount of urea are increased during the final days of an attack.

When a patient falls suddenly into hysterical slumber, or when

he is found asleep, the antecedents of the case being unknown, it is not always easy to recognize the nature of the sleep from its external characters alone. Although the hysterical sleep is a calm one, it may be accompanied by the phenomenon of apoplectic stertor and even by actual snoring. Ordinarily, however, there are no notable modifications of temperature, while in the comatose conditions due to organic lesions these modifications are frequent and well known. The contractures which accompany hysterical sleep rarely affect the systemic localization belonging to those which occur with organic lesions; and the same is true of the hysterical paralyses occurring in this form. Hysterical slumber may be simulated by certain cataleptoid states occurring in the melancholic insane in states of apparent stupidity associated with terrifying hallucinations. Hysterical slumber differs in hardly any respect, except in its spontaneity, from the hypnotic sleep; it is accompanied usually by the same abolition of conscience and may consequently favor the commission of the same crimes. The simulation of a sleeping attack by an hysterical subject is naturally very difficult to detect.

Demoniac Attacks.—At other times the hysterical attack is constituted solely by the contortions of the stage of extensive movements. One of the varieties of this form of attack is the demoniac attack of Charcot, in which we may see the most disorderly and strange movements and those of exceeding violence, accompanied by piercing shrieks, provoked by the painful contractures or by horrifying hallucinations. The demoniac attacks, in which we may often find in miniature all the stages of the grand attack, are generally of long duration, sometimes assuming the form of the status hystericus. The demoniac attacks are rather frequent in men, and they probably occupied an important place in the convulsive epidemics of the Middle Ages.

Rhythmical Spasms.—Comparable to the convulsions of the grand attack are the contortions, the rhythmical spasms, which we see figuring under the contagious form in the epidemics of the Middle Ages; saltatory chorea, epilepsy, and hysteria, which Germain Sée distinguished from Sydenham's chorea and called by the name of rhythmical chorea. Pitres has given us a specially good description of hysterical rhythmical spasms. These are convulsions, usually of sudden onset and without loss of consciousness, which recur at equal intervals and with a uniform cadence. They may be localized to one group of muscles and cause a very simple movement of flexion, extension, or rotation of the head or an extremity—rotatory chorea; or they may be more complex and systematized, simulating a professional movement—malleation chorea (*chorée malléatoire*).

These rhythmical spasms may occur alone, but they are also seen in the stage of extensive movements of the grand attack, in which they figure under the form of alternate movements of flexion and extension of the trunk, constituting veritable rhythmical bows. The attacks of rhythmical chorea may be provoked, like the other attacks, by physical shock or emotions; often they are excited by imitation, and this is especially true of saltatory chorea. The attacks may be repeated frequently or at longer or shorter intervals under the form of a continued series; sometimes the chorea is chronic from the first and continues for months or years or may persist even into old age (Charcot). Sometimes the hysterical tics become permanent, presenting paroxysmal exaggerations from time to time. Whatever may be the form of the hysterical rhythmical spasms, they have a great tendency to recur. Even when the spasms occur alone we may easily recognize their hysterical character by noting the presence of stigmata or by a history of other convulsive attacks having occurred in the same individual.

We may also observe in hysterical objects arhythmical spasms, a true arhythimcal chorea,[22] simulating Sydenham's chorea so closely as even to be mistaken for it.

The diagnosis is the more difficult because there is often, in girls suffering from chorea, an ovarian sensitiveness on the side most affected. Sydenham's chorea may have a sudden onset and appear, without rheumatic symptoms, after some mental shock, in all respects like hysterical spasms; it may also be unilateral; it is liable to recur; and it is frequent at the age of puberty. On the other hand, chorea, like the spasmodic manifestations of hysteria, is especially influenced by conditions which change and improve the nutrition. It seems then to be difficult to differentiate ordinary chorea and certain forms of hysteria, and although we may admit with Charcot that chorea in hysterical patients is merely a coincidence, we can nevertheless not deny that the two affections may grow in a common soil.

Coördinate spasms are not peculiar to hysteria, for we find them also in the affection known as myriachit, latah, or the jumping malady, which occurs most commonly in children and adolescents under the form of sudden muscular startings, often accompanied by the utterance of words under the form of echolalia or of coprolalia; these movements are rarely as regular as those of rhythmical chorea. The jumping malady presents none of the permanent stigmata of hysteria, and it generally is of chronic form from the beginning.

The electric chorea of Henning, Henoch, and Bergeron has been rightly grouped by Pitres among the rhythmical spasms of hysteria. The electric chorea of Dubini is an affection which begins with pains in the head, back of the neck, and lumbar regions, followed by twitch-

ings analogous to those produced by electricity. The affection rapidly assumes a grave aspect and terminates most frequently in death (Bianchi); it has therefore nothing to do with the spasms which we are now studying.

Paramyoclonus multiplex, which is characterized by the fact that percussion over the tendons invariably causes spasms, presents none of the permanent stigmata of hysteria, but it has nevertheless been regarded by some writers as related to hysteria.

Catalepsy.—The stage of emotional attitudes is often complicated by catalepsy; this condition may be observed in connection with other syndromes than those of the hysterical series, yet it is of frequent occurrence in attacks of hystero-epilepsy, in men as well as in women. Like most of the other episodes of the grand attack, catalepsy or ecstasy or rapture may occupy a predominating place in the paroxysm, or may even exist almost alone, though this is not common. The duration of the cataleptic state is very variable; when it forms a part of a major attack it is generally of short duration; but when it occurs alone it may last several hours or even several days, and may constitute a sort of status. Catalepsy is characterized by the possibility that the patient may assume spontaneously or may be placed mechanically in attitudes which remain fixed, although the respiration and the circulation of the subject betray a putting forth of not the least effort. The attitude of the patient is accompanied often by a corresponding expression of the face, so that in catalepsy and in spontaneous ecstasy the attitude would appear to be in relation with an emotional state provoked by an intense sensorial hallucination. The fixity of the attitude is not absolute, for the limbs finally obey the laws of gravity, and at the end of a certain period, which hardly exceeds the limit of effort of a strong man, the patient falls again into an attitude of repose. But this period of repose may occupy but a very brief interval between two cataleptic attacks so that we may observe a cataleptic condition without a return to consciousness, a sort of status catalepticus.

The diagnosis of hysterical catalepsy rests upon the presence or absence of other hysterical symptoms. The prognosis of the cataleptic attacks is the same as that of the attacks of sleeping; there is very little danger as regards life, but there is a great tendency to recurrence and the attacks may be of very long duration. The rare cases in which death has been attributed to hysterical catalepsy are as doubtful as are those in which death has been supposed to result from sleeping attacks. Like these also the cataleptic attacks cease spontaneously or under the influence of irritation of an hysterogenic zone or simply in consequence of an intense mental impression.

Delirium.—The delirium of the fourth stage of the hystero-epileptic attack may occur in the form of a single paroxysm or in that of a status; it may assume the most varied forms; and it may occur in both sexes and as well in children as in adults. Indeed, as was noted by Briquet, the paroxysms of delirium are more frequent in children, and in them they are often the first manifestation of hysteria and its only manifestation, for at that age the stigmata are often wanting. The attacks of hysterical delirium may occur under the maniacal or the melancholic form, and as a rule do not differ symptomatically from ordinary mania or melancholia. The differential diagnosis can be made only by the observation of other hysterical troubles and sometimes by the course of the affection. The maniacal access which does not occur as a sequel of the delirious stage of the attack may arise suddenly in consequence of a moral or physical shock. Sometimes it presents interruptions appearing suddenly and lasting a variable period. It may also terminate suddenly. These are, however, not characteristic features of hysterical delirium. The exuberance of the language, the variety of the gesticulations, the intensity of the hallucinations, the frequency of the representations of animals or of a change of personality so characteristic of epidemics of delirium, the transformation into animals of various kinds, are all signs pointing to a probability only, upon which too great stress should not be laid. The duration of the delirium may vary from a few hours to several months, and it is subject to relapses which may bear a relation in time to the menstrual epochs.

Pitres has described a form of delirium characterized by troubles of memory and of personality to which he has given the name of ecmnesia or partial amnesia, and in which the recollection of events occurring anterior to a certain period is perfectly preserved, while the remembrance of what has happened after this period is entirely lost. It results from this loss of memory that the patient reasons and acts as he would be presumed to reason or act in his childhood or adolescent period, according to the duration of the period of his life the memory of which has been lost.

Like the other abnormal paroxysms, the attack may terminate suddenly without any disturbance or following a convulsive attack. In general the restoration to health is complete. A knowledge of the previous history and the presence of permanent stigmata enable us to differentiate attacks of hysterical delirium from the delirious fits of degenerates, from acute attacks of ordinary mania, and from certain forms of toxic delirium, such as those caused by belladonna, hasheesh, and especially alcohol; alcoholic delirium, indeed, has a number of characters in common with the delirium of hysterical

patients, such as the terrifying hallucinations of sight, professional preoccupations, and a certain yielding to the influence of suggestion. These intoxications usually present certain somatic characteristics which enable them to be recognized; but we must not forget that they are capable of exciting hysterical phenomena.

Somnambulism.—Related to the attacks of sleep are the somnambulistic attacks which, whether spontaneous or provoked, are associated with hysteria. Notwithstanding the assertions of the school of Nancy, I am convinced that the somnambulistic phenomena susceptible of somatic characteristics occur only in subjects with hysteria or a neurotic taint—conditions which are distinguished only theoretically. Finally, the hysterical nature of spontaneous somnambulism, in children as well as in adults, has been well shown by the observations of Charcot, Richer, Chambard, Gilles de la Tourette, and Guinon.

Spontaneous somnambulism is a state in which the subject executes the actions of ordinary life, but remains without reaction to the stimuli which would excite these actions in the waking period. It may be diurnal or nocturnal. The attack of somnambulism, like the attack of delirium, may follow a convulsion or appear suddenly during the night, while the patient is in normal slumber, or during the day under a narcoleptic form. It is not very uncommon to find somnambulism constituting a more or less durable episode of the grand attack. During the somnambulistic attack, whatever may be the apparent lucidity of the acts, the general sensibility appears to be abolished; the patient reacts neither to contact nor to traumatisms which are capable of causing pain; upon awakening he has no knowledge of the wounds which he may have given himself.

During somnambulism the eyes may be open or half closed, the regard is fixed and without expression, the pupil is immobile. Vision is not abolished in the proper sense of the word; the patients may avoid obstacles, read, etc., but the objects which are foreign to their actual preoccupation do not appear to attract their attention. Just as they see only what they look at, so somnambulists hear only what they listen to. The senses, however, are not completely unresponsive to excitations, for we may see irritation of smell and of taste, as well as those of hearing and of sight, provoke various hallucinations (Guinon).

The psychical exaltation which we sometimes observe may be explained by the systematization of the attention and the narrowing of the capacity of consciousness which give rise to a more or less exclusive monoideaism. There results from this psychical state a considerable modification of personality, a second state which is accom-

panied occasionally by evidence of correlative changes in the physical condition consisting sometimes in the disappearance of the permanent hysterical symptoms (contractures, paralyses, spasms, etc.). This systematization of the attention plays an important rôle in the security of the movements in the most critical positions which may scarcely inspire the patient with fear. As soon as the attack of somnambulism has ceased the memory of it is effaced; but in subsequent attacks the patient may remember what has taken place in previous ones; one might say that it was a dream in several acts. The duration of the attacks of somnambulism is extremely variable; when they are prolonged so as to constitute a status, the second state may equal in duration the first, so that the personality of the subject may be doubled, divided into two periods of equal extent. The second state may sometimes present so exactly the appearances of the first or waking state that it has received the name of *vigilambulism*. These prolonged second states are met with in men as well as in women.

The phenomena which have been described in recent years under the name of *ambulatory automatism* may, when they are manifested in hysterical subjects, be regarded as attacks of somnambulism. It is an impulsive vagabondage which hardly differs in itself from the ambulatory automatism of epileptics. In the absence of the stigmata of hysteria (which are found in a measure also in epileptics) and of the characteristic paroxysms, the diagnosis may be reserved. The loss of memory is not constant, and it may be observed also in epileptics; the results of treatment are not always to be relied upon in diagnosis, for epilepsy is not constantly benefited by the bromides. Charcot thought that the manifestations of violence were characteristic of epilepsy, but they are sometimes wanting in epileptics and may be present in hysterical subjects. The migrations of the insane are characterized by the absence of stigmata and of convulsions, and by the partial or total persistence of consciousness; however, the insane wanderers, dromomaniacs, often present neurasthenic symptoms which are distinguished from hysterical symptoms rather in nosology than by their nature. Migrations are observed also in those suffering from the delirium of persecution, who find in the change of domicile a relief to their insane preoccupations, in some melancholics, in dipsomaniacs, and in those suffering from general paralysis; in these cases, however, it is comparatively easy, from the evident symptoms, to eliminate hysteria.

Psychical Disturbances.—Is there an hysterical insanity? The study of a large number of facts has not led me to modify the opinion which I expressed twelve years ago that there is not such a form.[24] The psychical manifestations attributed to hysteria must be divided

into two distinct groups. Those of one group form but an episode in the attack of hystero-epilepsy, and in a delirium, which varies according to the individual, but which is always the same in the sense that it has a fixed chronological place in the attack from which it is isolated rather in appearance than in reality. This delirium is the sole psychical manifestation which belongs legitimately to hysteria. There is nothing characteristic in the other troubles which, under the appearance of one or the other form of insanity, are seen in connection with the permanent symptoms of hysteria; there is no necessary relation between the insanity and the hysteria, they are simply two troubles combined in one individual. The mental condition of an hysterical subject in the intervals of an attack has no necessary connection with the convulsive neurosis; when we see a condition of folly, a true moral insanity, in hysterical subjects, we are not to regard it as belonging to the hysteria, for it may often remain after the hysterical symptoms have disappeared finally. It is true that the disturbances of sensibility in the hysterical may engender disorder in the psychical functions, but this does not constitute actual insanity.

Amnesia.—We have seen several times that the boundary line between hysteria and epilepsy is not very distinctly drawn. There is less reason to be astonished at this, since the characteristics of all neuroses are at bottom ill defined, and following hysterical attacks, convulsive, delirious, or other, we observe phenomena similar to those seen after an epileptic attack and, in general, after all nervous shocks. And first, we have to note retroactive amnesia embracing not only the period of the attack but also a certain length of time before it; for example, the patient may be ignorant of the circumstances which have provoked the paroxysm or of other equally important events. With this loss of retentivity which follows certain paroxysms there coincides sometimes a loss of receptivity, which is more or less persistent and which results in the prolongation of the amnesia for a certain period following the attack.[1] It is to this diminution of receptivity, which constitutes a sort of period of apathy, that the paradoxical name has been given of retroanterograde amnesia. This phenomenon may exist also as a consequence of a shock, and was described many years ago by Brodie.[2]

Trophic Disorders.

Hysterical troubles do not consist solely in disturbances of sensibility and motility, for the nutrition is also notably affected. Although, as we have seen, the modifications of the urine are not constant and cannot therefore furnish anything pathognomonic to assist

us in the diagnosis, nevertheless they are very frequent. But the nutritive changes are manifested by divers vasomotor troubles, which, through the mechanism of congestive œdema to which succeeds anæmic œdema, may explain a great number of morbid phenomena and especially those which show themselves on the skin, such as redness, œdema, dermographism, pemphigus,[35] ecchymoses, and hemorrhages.

Dermatoses.—Spontaneous erythemas and urticarias are seen especially in hysterical subjects as sequelæ of an attack; but these patients also present a special aptitude for artificial erythemas and urticarias excited by local irritation or emotions. We often also in these patients observe a special aptitude to vasomotor reactions in general; they are especially subject to morbid blushing and to hot flashes. Sometimes the attacks are announced by troubles which recall the different stages of Raynaud's disease. But the vasomotor troubles may be encountered at other times than during the convulsive paroxysms, particularly on awaking or during the night. These vasomotor phenomena have dependent upon them also certain secretory troubles, especially ptyalism, and sweating localized more or less strictly to one spot, to one side of the body, or to the extremities. In some patients these sweats of the extremities, which may be exceedingly troublesome, are provoked by emotions or by intellectual labor.

Among the cutaneous manifestations of these vasomotor disturbances we have to note the localized œdemas of the skin and especially the so-called spontaneous urticaria and dermographism. The urticaria is sometimes preceded by a cutaneous hyperæmia accompanied by itching which provokes scratching and this in turn is quickly followed by a characteristic eruption; this form has a close analogy with dermographism or cutaneous œdema caused by pressure and various irritants of the skin. The urticaria may, however, occur spontaneously. It is generally ephemeral, but in subjects who may have trophic troubles excited by suggestion, the urticaria may have a remarkable persistence. Acute œdema of the skin (Quincke), angioneurotic œdema of Strübing, may exist without pruritus; it is usually located on the face, and may occur in hysterical subjects.

In connection with the urticaria we have to mention also cutaneous affections characterized by eruptions of vesicles, bullæ, or pustules. Pemphigus has been rather frequently observed, herpes more rarely. These eruptions are quite frequently surrounded by a well-marked erythematous zone. Sometimes the herpetic eruptions take the form of zona; in one case which I have observed the herpes zoster extended along an intercostal space starting from a spinal hysterogenic zone. Sometimes the zoster assumes the gangrenous form (Kaposi). A few cases of eczema have been reported. The develop-

ment of these cutaneous lesions may be in relation to the hysterical attacks; most commonly they appear after an attack, but they may precede it. Hallopeau and Larat have reported cases of dyschromia, achromatous patches surrounded by strongly pigmented papular zones, which they regarded as hysterical. We may mention also the eczematiform dermatitis of Frèche and Parmentier.

Vasomotor paralysis of the extremities, or erythromelalgia, has several times been observed in hysterical subjects, as has also Raynaud's disease, the course of which is in some cases marked by oscillations corresponding to the hysterical manifestations. But we see also in hysterical subjects gangrenous affections of the skin which in general coincide with other hysterical troubles; they occur under two forms, as patches appearing successively in the same region, and as disseminated patches. Sometimes these gangrenous troubles are caused by slight traumatisms, sometimes they are spontaneous and cannot be attributed to any external cause. Spontaneous gangrene is sometimes preceded by very severe pains in the part.[36] The cicatrization of these gangrenous patches is usually slow. Strümpell had an hysterical patient who simulated localized gangrene of the skin by applying pieces of caustic potash.

The Hair and Nails.—In connection with the trophic troubles of the skin we may mention those of the appendages of the skin, the hair and nails. Several years ago I observed a splitting of the ends of the hairs following an hysterical attack.[37] This phenomenon may perhaps be related to the drying of the skin which is so frequent in hysterical subjects. Fabre has observed canities and alopecia following emotions in hysterical subjects. On the other hand, Lejamptel[38] has seen an exaggerated growth of the hair and nails in hysterical paralysis. Pitres notes the possibility of a spontaneous falling of the nails analogous to that which occurs in ataxic subjects. White spots in the nails are quite common.

We must add to these trophic troubles of the skin the general dryness of the integuments, which is very common and may induce a modification of the electrical tension of the body characterized by the production of sparks in the hair upon the least contact with the comb, an attraction of light bodies by the skin, etc.[4] These manifestations, which increase under the influence of powerful emotions, peripheral irritations, etc., are sometimes most marked on the hemianæsthetic side. They disappear during damp weather. Their increase or diminution is accompanied by changes in character of the individual who is in a state of excitement when the skin is dry and the tension is strong, and is depressed during damp weather when the tension is relaxed.

Hutchinson has remarked that the dryness of the skin in hysterical subjects may lead to the formation of transverse fissures of the palms of the hands or soles of the feet, which may be rebellious to treatment and painful.[29] The patient in whom I observed the electrical phenomena just described had multiple cutaneous fissures in the continuity of the members.

Œdema.—The vasomotor troubles which give rise to these cutaneous alterations in hysterical subjects are also the cause of deeper lesions, especially of the subcutaneous cellular tissue which is often the seat of œdemas of variable extent and duration, such as blue or congestive œdema and white or anæmic œdema. Hysterical œdema was observed by Sydenham who noted its two chief characteristics, namely, an absence of permanent depressibility and a morning predominance. Brodie compares it to an urticarial wheal of great extent, and remarks that when it coincides with an articular affection it often involves the entire limb. Charcot especially has given an extended description of hysterical œdema. It may be encountered in the two sexes, but almost exclusively in adults. It only exceptionally occurs as an isolated phenomenon in the region which it occupies, but most commonly coexists with an arthralgia, a paralysis, or a contracture, and is accompanied usually by anæsthesia. Most commonly it is unilateral and localized, but it may extend over an entire side, over the breast, the loin, the thorax; in exceptional cases it is bilateral and generalized.

The color of the œdematous parts varies greatly in different individuals and also in the same individual under the influence of the hysterical attacks; it varies from a dull white to a slaty blue, passing through pink, red, and blue. It is a hard œdema, not pitting on pressure of the finger, fading off gradually at the periphery. It is more or less extensive, sometimes limited to a very circumscribed region, and presenting the appearance of a huge urticarial wheal. At other times it extends over an entire member and may even be generalized. It varies also greatly in volume. In the case of a white œdema the temperature remains normal; in pink or red œdema we find occasionally a slight elevation of temperature, while in blue œdema the temperature is subnormal. Instead of anæsthesia and analgesia, which are the rule, we sometimes find in the affected region spontaneous pains and tenderness on pressure—a dysæsthesia. The œdema which accompanies motor troubles may follow their course strictly, appearing and disappearing coincidently with them; it may, however, precede these disturbances and remain after their disappearance. Notwithstanding its permanent character, hysterical œdema is an essentially variable phenomenon, increasing or diminishing in

consequence of an infinity of causes, physical and moral. All conditions of depression are capable of exaggerating it, as are also sadness, fatigue, cold, darkness; the opposite conditions, on the other hand, cause a temporary diminution of the œdema. It is usually influenced by the menstrual period, at which time it is most frequently increased.

Generalized white œdema may resemble phlegmasia alba dolens; a pink circumscribed œdema has been mistaken for a phlegmon and has been incised, giving exit to black blood only; blue œdema might resemble Raynaud's disease, but it is not symmetrical, does not affect the nose or ears, and does not appear gradually but suddenly. Hysteria with blue œdema and thermoanæsthesia may simulate syringomyelia (Charcot).

In connection with oscillating œdemas we must mention intermittent hydrarthroses, which, although not belonging exclusively to hysteria, are sometimes seen in connection with it, appearing in a child affected with other hysterical troubles and undergoing remissions and exaggerations as a result of the same influences as the hysterical accidents. Morel has recently reported a case of hysterical epididymitis occurring in paroxysms coincident with those of nervous œdema.

Hemorrhages.—While on the subject of these œdemas we may conveniently study the hemorrhages which are not uncommon in hysterical subjects. These hemorrhages may take place on the surface of the skin or mucous membranes or in the substance of the cellular tissue and especially in the subcutaneous cellular tissue. Spontaneous ecchymoses are rather frequent, and as they give rise to no sensation they may pass unperceived when they are situated in a part which the patient cannot see. The knowledge of the possible existence of these ecchymoses is exceedingly important from a medico-legal point of view, for they might readily be mistaken for the results of traumatism. They often coincide with other forms of hemorrhage or with œdema. Their appearance is sometimes accompanied by pain, local dartings, and they are then not unlike the ecchymoses which sometimes accompany the lightning pains of locomotor ataxia or of neurasthenia. At other times they come on after dreams or hallucinations of blows or violence of some kind; they then constitute the *stigmata diaboli* which have held an important place in the history of possessions and also in certain celebrated legal processes. The ecchymoses may be produced on any part of the body, but in some patients they appear always in the same region (stigmata of crucifixion, etc.). Sometimes they appear under the mucous membranes of the eyelids or ball of the eye, mouth, etc.

The ecchymoses are quite frequently accompanied by true hemorrhages of greater or less abundance. Moreover, in patients subject to spontaneous ecchymoses, the slightest traumatisms may provoke subcutaneous hemorrhages or external bleeding. The hemorrhages may manifest themselves under the form of epistaxis, otorrhagia, bloody tears, or bloody sweat. At other times the blood issues through a solution of continuity of the skin on the dorsal surfaces of the hands or feet, or from the forehead. At the point where the hemorrhage occurs we often observe a swelling of the integument, ampullæ which complete the picture of the stigmata; sometimes there is a tumefaction over a more or less extensive surface, and generally there is at least a slight œdema of the skin which is sensitive, cold, and a little red. The blood always has the characters of arterial blood. Whether or not it has been preceded by pain and accompanied by other troubles coinciding with the convulsive paroxysms, the hemorrhage is exceedingly variable in amount and duration. Relapses are frequent and are often marked by a remarkable periodicity. The mucous hemorrhages which we have already referred to, as well as hæmatemesis and hæmoptysis, often have this same periodicity without being in any way related to the menstrual flow which, in many cases, continues regularly and in normal amount.

Hysterical Breast.—Following a consideration of the œdemas and hemorrhages connected with vasomotor troubles comes naturally that of the hysterical breast, the nature of which was well recognized by Willis, Pomme, Watson, Astley Cooper, and Brodie. The mastodynia may be determined by a traumatism, by some lesion of the genital organs, or by a sort of suggestion when some acquaintance suffers from a tumor of the breast; but frequently the cause remains obscure. The neuralgia appears most often to coexist with an increase of volume, although this enlargement is not always readily appreciable. We have then a dysæsthesia characterized by spontaneous pains and by an exquisite sensibility to the slightest contact. This dysæsthesia may extend over the entire corresponding side of the body and it generally coincides with a sensory anæsthesia. Furthermore the exaggerated sensibility of the skin and the pain coincide with a diminution of tactile sensibility in all its forms. The permanent dysæsthesia is exaggerated under the influence of the emotions, of the menstrual periods, of certain peripheral irritations, and of certain forms of food and drink. A shock or pressure on an hysterogenic zone may bring on a paroxysm which sometimes coincides with a convulsive manifestation.

Ordinarily the pain coincides with a swelling of the breast and sometimes with a redness of the integument. In a woman whom I

have had the opportunity of observing, both breasts were perfectly symmetrical in the intervals of the attack and the skin presented no change in color. While I was examining her, her companion made an offensive remark, and under this influence, at the same time as the skin reddened, the left breast, which was the seat of the trouble, became marbled over with little red spots, like a scarlatiniform rash, the patches of which soon coalesced, forming a uniform redness which passed a little beyond the limits of the breasts, but did not spread along the course of the nerves. At the same time as the skin grew red, the breast became uniformly swollen, and erection of the nipple occurred. The entire region became the seat of a smarting sensation with pricking of the skin and dartings into the mammary gland which became heavy. All these phenomena reached their height within a minute.[10] The congestion of the skin may be wanting, but it is sometimes so intense as to assume a violet color. Sometimes the tumefaction disappears completely in the interval of the attacks, but when the latter recur frequently and have existed for a considerable period, the swelling becomes permanent.

Palpation, which is usually impossible during the paroxysm because of the pain, sometimes reveals to us during the interval the presence of distinctly circumscribed tumors (Fowler) which are more or less movable, together with engorgement of the milk ducts. Certain cases of this nature cited by Fowler, in which ablation had been advised, were cured by psychical treatment. Gilles de la Tourette cites several cases in which amputation of the breast was practised without result.

Muscular Atrophy.—Our knowledge of muscular atrophy in hysteria is of recent date; a pupil of Seeligmüller had noticed it in 1884, but it has been more especially studied by Babinski who states that it has the following characters: diminution of volume in variable degree and sometimes very rapid, absence of fibrillary twitching, normal idiomuscular excitability; diminution of electrical irritability in proportion to the degree of atrophy, but without the reaction of degeneration; recovery as rapid, sometimes, as the onset. These characters, however, are not absolute. These muscular atrophies are seen most frequently in men; they are of quite common occurrence following a traumatism, and coincide usually with a paralysis or contracture. This coincidence is the rule, yet cases have been observed in which the atrophy was on the side opposite the hemiplegia (Charcot). It may be generalized in the paralyzed or contractured member; as a rule it is preceded by pricking sensations, formication, and sometimes fibrillary twitching (Gilles de la Tourette). It affects the small muscles of the peripheral parts as well as the large muscles of

the limbs. Sometimes it is limited to certain groups of muscles, such as those of the shoulders, arms, or hands. Simple hysterical atrophy is ordinarily accompanied not only by paralysis or contracture, but also by troubles of sensation; this constitutes an important characteristic, but which may be met with also in syringomyelia. The duration of the atrophy is exceedingly variable; it may last for months or years, and, when the paralysis or the contracture gets well more or less rapidly, a certain length of time always elapses before the muscles regain their normal volume.

This muscular atrophy may coincide with trophic lesions of the bones and of the skin; the fingers become tapering and their integument becomes thin and assumes the appearance of glossy skin. These troubles may appear with great rapidity and disappear just as suddenly. The presence of hysterical stigmata enables us to make a correct diagnosis.

Pitres has noted a spontaneous falling of the teeth in an hysteri cal patient who had had repeated attacks of paralysis.

Following long immobility in consequence of paralysis and especially of contracture, we may find fibrotendinous retractions which may necessitate surgical intervention; this is usually efficacious. The cause of these ankyloses appears to reside in the tendons and their sheaths and the surrounding tissues rather than in the articulations themselves.

Apart from any paralysis or contracture we sometimes observe in the tendinous sheaths and synovial bursæ crepitation and loud crackings, which may be increased and cultivated" and which appear to be due rather to a peculiar tension of the muscles than to any trophic lesions in the tendon sheaths.

Pyrexia.

Hysterical fever should be regarded as a part of the paroxysms much more than as a trophic trouble, in which category it is apt to be placed; its chief characteristic, indeed, is the very fact that it is not accompanied by any marked degree of emaciation.

The older writers, Baillou, Rivière, Tissot, and Robert Whytt, admitted the existence of a nervous fever. Pomme recognized an hysterical fever which was perhaps a pseudo-fever, and which did not rest upon facts sufficiently authenticated to withstand the doctrine of Broussais, with which disappeared nervous and hysterical fever. Beau, however, admits the existence of a false typhoid fever of nervous origin, and with Briquet hysterical fever was again recognized. However, any facts adduced before the introduction of the clinical thermometer were hardly certain, and Pinard again, in 1883, denied

the existence of a true hysterical fever. He recognized an hysterical pseudo-fever, without elevation of temperature, characterized by a temporary acceleration of the pulse, which he regarded as related to the permanent tachycardia of exophthalmic goitre. But the observations of Debove, Barié, Fabre, Auffleck, Bressler, Hanot and Boix, Vizzioli, Spoto, and others[12] have put beyond doubt the existence of a true pyrexia of hysterical origin.

Hysterical pseudo-fever is the more frequent, but it has not the same importance; it is manifested under the continuous, intermittent, and remittent forms. It adds much less to the difficulties of diagnosis than does the true hysterical fever; the latter may appear alone or in connection with the other manifestations of the hysterical paroxysm or with a special complex of symptoms which simulate a visceral affection.

In simple hysterical fever the course of the temperature is extremely variable, being sometimes as elevated in the morning as in the evening, sometimes higher in the evening than in the morning; sometimes the high temperature is interrupted by subnormal temperature; it may be as low as 29° C. (84.2° F.). A maximum temperature much higher than would seem to be compatible with life has been observed. These hyperthermal attacks are terminated most commonly by a rapid fall of temperature. Occasionally the surface temperature is said to be unevenly distributed. The pulse and the amount of perspiration often bear no relation whatever to the degree of temperature. The fever appears often suddenly, following the receipt of some mental shock, and it may disappear in the same way and from the same cause. Hysterical fever may coincide with the attacks of convulsions, which do not arrest it. This absence of antagonism between the spasm and the fever is not an exclusive characteristic of hysterical fever; I have published a case in which frequent convulsive attacks occurred during the course of an acute articular rheumatism accompanied by heart complications and fever. Simple hysterical fever may occur under a remittent or an intermittent form; the latter may present the picture of the three stages of a malarial fever. When hysterical fever is accompanied by symptoms apparently pointing to the existence of visceral lesions, it may assume various forms. The false typhoid fever described by Beau has been found to be hysterical in nature by Rigal, Bertoye, and Hanot and Boix. Other forms which have been noted are the pseudomeningitic, dyspnœic, and peritonitic, reproducing the symptomatology of acute meningitis, grave pulmonary affections, and peritonitis. Indeed, hysteria plays an important rôle in the etiology of pseudoperitonitis (the peritonism of Gubler) and in pseudomeningitis (meningism of

Dupré). This latter is particularly interesting, since it has been encountered in children.[43] The presence in the neighborhood of another patient suffering from a real visceral affection seems to play some part in the development of these forms of hysterical fever.

The principal characteristic of hysterical fever is the absence of emaciation. There is no doubt that the examination of the urine will be of great assistance in the diagnosis of hysterical fever, if it shows a diminution of fixed residue, or urea, and of phosphoric acid.

Paralyses and Contractures.

A great number of hysterical subjects present in what is their normal condition, in the intervals of the attacks, a certain degree of motor weakness, usually more marked on one side and most frequently the left. This weakness coincides ordinarily with anæsthesia or some of the various troubles of sensibility. Or again, in place of a simple weakness of the organs of motion, we may sometimes observe functional anomalies, a tendency to rigidity, to contracture, or to tremor. These germs of paralysis and of contracture may become developed in a great number of conditions, usually those associated with a depressed state of the organism.

We may find references to hysterical paralysis and contracture in the oldest writers, but their scientific study is of recent date. It will suffice to mention the names of Brodie, Piorry, Macario, Laycock, Gendrin, Todd, Türck, and Romberg, who have made interesting contributions to the literature of the subject; after them came Briquet, and finally Charcot. For a long time it was held that these paralyses and contractures formed in a measure appendices of the attacks, but in reality they hardly have anything to do with the attacks in more than half of the cases and perhaps more particularly with those which have been called apoplectiform or lethargic. The emotions, if intense or long continued, may produce these motor troubles. Infections and intoxications may produce the same effects, acting as causes of depression, and so may cold. Fatigue may have the same effect (paralysis by exhaustion), and it appears to be capable of acting even when it is but the result of a dream; I have seen a paraplegia which seemed to be the consequence of a prolonged dream. Traumatisms, shocks of all kinds, local affections, and rheumatism, all appear to play an important rôle in the production of these paralyses and contractures. Charcot attributes a preponderating influence to the imagination in the production of traumatic paralyses, and he has given the name of period of preparation, of meditation, to the period of time which separates the receipt of the shock from the appearance of the paralytic symptoms. This period

may often fail, however, the paralysis manifesting itself immediately; and furthermore, the phenomena of shock which are often seen in the lower animals show that the intervention of the imagination is not indispensable. The effect of physical or moral shock may be more simply explained as mere fatigue; and most so-called psychic paralyses (dependent on idea, Russell Reynolds; durch Einbildung, Erb) are preceded by conditions of exhaustion and of depressing emotions.

It is not only the violent and depressing excitations which are capable of causing hysterical paralyses; there are paralyses by in-irritation, to revive an expression that Erasmus Darwin employed in another sense. In consequence of the absence of physiological irritations, we may see the appearance of transitory paralyses which are instructive from the point of view of the pathogenesis of hysterical paralyses; the night palsy of Weir Mitchell would often belong in this category.

The exciting cause of paralysis or contracture has some relation to the localization of the trouble. The paralysis which follows an apoplectiform attack is often hemiplegic, that which follows a traumatism is usually most marked in the injured limb; and it is the same with that which follows excessive labor, for it is most pronounced in the part in which the fatigue is most felt. The paraplegic form is not uncommon after moral shocks. In a general way it may be said that it is the side which is amyosthenic in the habitual state that is most liable to be affected; it is not surprising, therefore, that hysterical paralyses and contractures are most frequent on the left side.

The paralyses and contractures may be found in both sexes; they are rare in the aged and in children below the age of ten years. The varieties of paralysis and contracture are not equally frequent; the hemiplegic form is the one which is most often encountered—in about half of the cases, and usually on the left side; the paraplegic form is seen in only about one-sixth of all cases; the localized forms are exceedingly variable, the different ones taken by themselves are rather uncommon, although altogether these form about one-third of all the cases.

Paralyses.—The clinical study of hysterical paralyses has been particularly well done recently by Paul Richer.[4]

The onset of the paralysis may be slow or sudden, even instantaneous. Sometimes it is preceded by formication, numbness, or pain. As a rule it is incomplete, and concerning this Richer has noted that even when both legs are affected and the patient is confined to the bed, a careful examination will show that the paralyzed members are capable of motion in some of their parts, although this power is, it

is true, very slight. The paralysis affects the antagonistic muscles equally. It is only rarely accompanied by nutritive disturbances, and the lowering of temperature, which has been noted, is very inconstant. It is not only voluntary movement which is affected in hysterical paralysis, but the muscular tonicity as well; this is well seen in the face where the wrinkles are effaced and the nostrils are flattened; the mouth is often drawn to one side. This loss of tonicity is not by any means peculiar to organic paralyses, as has been asserted. The paralyzed muscles have lost their electrical sensibility; their electrical contractility is preserved, but whatever may be the intensity of the current it is not felt. Sometimes, however, the electrical contractility is diminished or even abolished, and Richer observed a case in which the power of voluntary movement returned before the electrical contractility. Very often hysterical paralysis is accompanied by troubles of sensibility which are superposed upon it with a remarkable exactitude (Charcot). Sometimes, however, the disturbances of sensibility are much more extensive than the motor troubles and may involve an entire side, including the organs of special sense.

The cutaneous reflexes rarely persist, even when the sensibility is not abolished; the tendon reflexes are, on the contrary, most commonly exaggerated. We can often excite even an epileptoid tremor; the trouble is a sort of spasmodic paralysis and is readily transformed into contracture. It is very exceptional to observe a diminution or abolition of the tendon reflexes, and in such exceptional cases a contracture is hardly to be feared; but in the course of a relaxed paralysis with loss of reflexes, we may occasionally see the latter reappear and then become exaggerated, and in such case the opportunity for contracture returns. One of the characteristics of hysterical paralysis is to vary in intensity under the influence of very slight causes or even without any appreciable cause whatever.

The paralysis is sometimes fixed and obstinately resists all treatment for years, while at other times it is not only variable in its intensity but changeable in its seat, passing from one side to the other, from one region to another, affecting successively an arm, a leg, the muscles of the larynx, the diaphragm, etc. Termination in recovery is the rule, and it may be gradual or sudden. The latter occurs especially as the consequence of some lively mental impression. A gradual recovery is sometimes announced by feelings of uneasiness in the limbs or by dreams of movement. In one patient, a woman, the diminution of the paralysis was first manifested by the ability to move in the bath. Very frequently the paralysis is transformed into a contracture, which may show itself suddenly following

a traumatism or a moral shock; more rarely the contracture is established gradually.

Astasia-abasia.—Charcot and Richer emphasized especially a motor trouble which holds an important place in the history of hysterical paralyses, the existence of which had been briefly referred to by several writers, especially by Jaccoud; in this the patients are able to make rather energetic movements while in the bed, but are incapable of standing erect or of walking. The affection has been studied considerably of recent years, and especially by Blocq, who has given it the name of astasia-abasia. Richer remarks that astasia and abasia most frequently coexist, but while abasia may exist alone astasia cannot exist without abasia; that is to say, it is possible that a patient may stand without being able to walk, but no case is known in which walking was possible and standing impossible. Astasia-abasia is regarded not as an incomplete paralysis, but as a systematic palsy of the movements necessary for walking and standing; its characteristic feature is held to be the integrity of movements when the patient is in bed. But absolute integrity of movements can be demonstrated only by measurements of energy and rapidity of motion, and thus far these measurements are wanting in cases of astasia-abasia. I have had occasion to make a dynamometric examination and a study of the reaction time both before and after recovery in hysterical subjects whose incomplete paraplegia recalled the characters of astasia-abasia; and as long as these troubles of walking and standing lasted, there was, while the patient was in bed, a diminution of energy of the different movements together with an increase of the reaction time.[45] In order to demonstrate the existence of systematization of a paralysis, it is absolutely necessary to prove the integrity of all movements not adapted to the special function in question. We shall return to the clinical history of this incomplete paralysis, which may be manifested in the upper extremities and affect specially the most delicate functions.

Contracture exists often in the latent condition (diathesis of contracture) in hysterical subjects, who have an exaltation of the tendon reflexes, epileptoid tremor, and peculiar electrical reactions which have been well described by Richer. Real contracture is easily provoked by various stimuli, but especially by shock and local irritations.

Permanent hysterical contracture may be accompanied by anæsthesia or dysæsthesia. Painless contracture is the more common; it may appear suddenly after the receipt of a traumatism or in consequence of some strong emotion; it is then sometimes the first manifestation of hysteria. At other times it is preceded by a weakness or numbness of the limb or by spasmodic symptoms, such as epileptoid

tremor, twitchings, etc. It may increase gradually or it may attain its maximum at once. The latter is the more usual mode of onset. The stiffness is considerable and is fixed, but it may sometimes be overcome by a slow, continued, and even traction. Whatever may be the attitude of the limbs, the antagonistic muscular groups are equally affected, although there must be some predominance of action on one side or the other which determines the attitude taken. While hysterical paralysis varies frequently in intensity, contracture is in general remarkably persistent and unvarying. There may be a momentary resolution during sleep, but in cases of some standing this resolution is usually incomplete. The sensibility is ordinarily affected just as it is in cases of paralysis; anæsthesia is the modification most commonly noted. Too often the contracture persists for months absolutely immobile, but sometimes it disappears suddenly and then as suddenly reappears—*contracture à répétition;* at other times it disappears from one region to reappear in another—*contracture erratique* (Richer). Its termination may be slow or rapid. The slow termination is effected by a diminution of the stiffness which is replaced by a paresis, which in turn disappears slowly; but the tendency to contracture often persists for a long time. The sudden termination is rather frequent as a consequence of emotions, of the so-called subversive treatment; but even in these cases we often see a rather marked paresis persist for some days, and the tendency to contracture still remains, showing itself sometimes for months by an epileptoid tremor.

Although usually painless, permanent contracture may sometimes be accompanied by shooting pains or very troublesome formication in the affected members. Occasionally these pains are so intense that the patient begs for an amputation of the limb. In one such case Charcot advised the stretching of the median nerve, and this was followed by a disappearance of the pains. Briquet has seen successful treatment of painful contracture by means of wet cups; opium in large doses may also be of service.

Sometimes the contracture is variable, becoming exaggerated through the influence of the attention or by manœuvres intended to overcome it. Instead of persisting constantly it may cease during sleep; again the attitudes may not obey the law of being in flexion for the upper extremity and in extension for the lower; and the troubles of sensation may also be wanting. Richer thinks that these modifications are characteristic of what he calls psychical contractures.

Complications of hysterical paralyses and contractures are seldom encountered; the paralyses are only exceptionally accompanied by

muscular atrophy, and the fibrotendinous retractions seen in connection with contractures are also very rare.

The paralysis may be hemiplegic, monoplegic, paraplegic, or quadriplegic, and the contractures may assume the same forms. Hemiplegia, either flaccid or rigid, is one of the most common forms of hysterical paralysis; in nearly three-quarters of the cases it is located on the left side. Hysterical hemiplegia follows and invades the same parts as hemianæsthesia and hemiamyosthenia, of which it seems to be an exaggeration. Hysterical hemiplegia may occur at all ages, but is most frequent in adults who are most exposed to the exciting causes of the affection.

The intensity of the paralysis varies greatly, from a slight paresis to a complete paralysis. Unlike what is usually seen in organic hemiplegia, the lower extremity is ordinarily the most affected in hysterical hemiplegia. When the motor paralysis is very pronounced, the troubles of sensibility are usually also more marked, and in such cases the special senses are often involved. The walk is quite characteristic. In organic hemiplegia the patient carries the trunk first over to the non-paralyzed side and rests all the weight of the body on the sound limb, then carries the paralyzed limb forward, making it describe the arc of a circle; the hysterical patient, on the contrary, drags the affected member after him, sweeping the ground with it. This walk of the hysterical paralytic is not strictly speaking characteristic, for it is the gait of flaccid hemiparaplegia.

The diagnosis of hysterical from organic hemiplegia may present certain difficulties. Sensitivo-sensorial anæsthesia does not exclude the possibility of an organic lesion, for these unilateral troubles of sensibility may be associated with a capsular lesion, and even concentric narrowing of the visual field. The preservation of the tendon reflexes in hysteria is a valuable sign, but it may be wanting. It is by a study of the other associated troubles and of the previous history of the case that we can usually arrive at a diagnosis.

The hemiplegic form of contracture is more rare than flaccid hemiplegia. The superior extremity is generally in flexion; the arm is in adduction, the forearm in supination is flexed at a right angle with the arm; the hand is also flexed. In the exceptional case of extension of the forearm, the hand is generally flexed in forced pronation, and the fingers are lightly flexed in the palm. In the lower extremity extension is the more frequent position, and is often accompanied by rotation inwards.

It has long been a matter of dispute whether or not facial paralysis exists in hysterical hemiplegia. While Todd, Althaus, Hasse, and Charcot regard it as rare and even doubtful, Briquet, Le Breton,

and Hélot hold that it really exists, even if it is not frequent. The observations of Brissaud and Marie and of Gilles de la Tourette have shown that in hysterical hemiplegia there may be a condition of spasmodic contraction, sometimes with twitchings of the muscles of the face on the opposite side, together with superposed anæsthesia or dysæsthesia. These facts have seemed to argue against the existence of facial paralysis, but the observations of Chantemesse and of Ballet (1890) have since established its existence without question. I had previously shown that, even in simple hysterical amyosthenia there is paresis of the tongue on the corresponding side.* An analogous paresis can be demonstrated in the lips and the masseter muscles. We may further note in hysterical amyosthenics that there is a retarded reaction time in the weakened muscles of the face and jaw. I have noted that the glossolabial contracture on the side opposite the paralysis of the members may be the consequence of a paresis of the face on the hemiplegic side. I have indeed found that in the induced paralysis of hysterical patients there is often an increase of motor energy on the opposite side. In one case I observed that as the hemiplegia became less, there was a diminution of force on the healthy side. This sort of balance of nervous action is not readily explicable. I may recall, however, that Claude Bernard has noted an analogous fact in the course of his studies on the sympathetic nerve: "At the same time that galvanization of the upper part of the sympathetic nerve reduces the temperature of the corresponding ear, we see the temperature of the other ear become elevated. This is a constant fact."

Facial paralysis and glossolabial spasm ordinarily coincide with the hemiplegia. Sometimes the face is contractured, while the corresponding limbs are flaccid. Sometimes the paralysis or the spasm coincides with a monoplegia; Charcot has observed a glossolabial spasm coincident with a paraplegia, the upper extremities being exempt. Paralysis and spasm may be observed alone as, for example, after a traumatism. Facial paralysis may occur under the tonic or the clonic form. I have seen clonic spasm coincide with a spasm, also clonic, of the sterno-cleido-mastoid muscle of the same side. Exceptionally the spasm, although more marked on one side, involves the entire face; at other times, on the contrary, it may be localized to a small number of muscles. The muscles innervated by the motor branch of the trigeminal may be affected like those supplied by the facial. By means of a special dynamometer and by chronography I have found that the contraction of the masseters is retarded and weakened in hysterical hemiamyosthenia; the masseter may also be alone affected by a spasm, as, for example, in consequence of a dental trouble.

Spasm affects most frequently the inferior facial and platysma myoid (glosso-labio-platysmal spasm of Gilles de la Tourette). The tongue is excessively deviated. When this spasm becomes very intense the domain of the superior facial is affected. Exceptionally the tongue or the orbicularis palpebrarum is alone affected.

Hysterical facial spasm can hardly be confounded with secondary contracture of the face in which disturbances of sensibility are usually wanting and which is hardly limited to the face. The condition is shown upon voluntary movement, just as is paralysis, which may pass unperceived in repose, being betrayed only by a slight relaxation of the features and especially by a flattening of the nares, which is seen frequently in simple amyosthenia. Neither in spasm nor paralysis are there any modifications of electrical reaction. Both the isolated paralyses and spasms of the face usually get well spontaneously within a few months, although cases have been observed in which they lasted for years.

Paralyses of the muscles of the neck, which exist to a certain degree in hysterical hemiplegia, may also be seen as isolated phenomena; Richer has reported a case of paralytic torticollis, but contracture is more frequent than paralysis, so that spasmodic torticollis is more frequently seen. This spasmodic torticollis may manifest itself in the intermittent (J. Voisin) or in the rhythmical (Richer) form.

In hysterical hemiamyosthenia I have observed a diminution of the diaphragm phenomenon of Litten.[41] This diminished excursion of the diaphragm seems to imply a weakness of this and of all the muscles of inspiration. No observations of isolated paralyses of the trunk muscles have been recorded except one of paralysis of the serratus magnus reported by Verhoogen. More commonly the muscles of the trunk are affected by contractures which may occasion deformities. Duret has described an hysterical kyphoscoliosis and after him many cases have been reported of hysterical curvatures of the spine, involving almost exclusively the lumbar region and accompanied ordinarily by a general rigidity of the trunk. The spinal curvature is generally one of large radius. When it is accompanied by local pain and especially by paraplegia it may be mistaken for Pott's disease. These curvatures, which disappear under the influence of general anæsthesia, may be attributed to contracture of the muscles of the deeper layer of the back, of the quadratus lumborum and psoas. These contractures are of variable duration and are apt to recur. Briquet has observed that contracture of the abdominal muscles on one side is also capable of producing deviations of the trunk.

Hysterical paralyses and contractures of the extremities are also observed in the localized or monoplegic form. It is traumatism which plays the most important part in the etiology of hysterical monoplegias; the upper extremity, which is the most exposed, is the most apt to be affected by these paralyses, which, for the same reason, are more frequent in men. It is especially in these traumatic monoplegias that Charcot has regarded the suggestion under the influence of pain as an important etiological factor. The psychical theory of paralysis is based on the fact that the motor troubles do not always immediately follow the shock, but are often preceded by a period of meditation or preparation. It may be objected to this theory that the physical effects of shock are not complete immediately after its intervention; we must distinguish also the different parts played by emotion and imagination.

The brachial monoplegias may be generalized or limited, complete or incomplete. Sometimes they are associated with a paretic condition of the lower extremity. The paralysis is accompanied by cutaneous anæsthesia, which affects a peculiar disposition; it does not follow the distribution of the nerve trunks but occurs in areas representing geometrical segments with well-defined boundaries, and superposed on the paralyzed muscles like a cuff, a sleeve, or a glove, according as the paralysis involves the forearm, the shoulder and arm, or the hand. The "mitten" anæsthesia coincides with a paralysis of the hand without involvement of the fingers, a form which is not very rare. All the forms of cutaneous sensibility—to pain, to temperature, and to electrical currents—as well as tactile sensibility, are abolished; and the same is true of the muscular and articular sense as well as the sensation of position. It may, however, happen that the tactile sensibility is preserved, while that of temperature is abolished, this so-called syringomyelic dissociation of sensibility is not in contradiction with the sometimes profound trophic disturbances which may be encountered in hysterical palsies.

The course of a brachial monoplegia is extremely variable. These paralyses are usually of short duration in children, but not so in adults, especially in men; cases occurring in the latter have been reported which lasted for twenty years. These monoplegias are furthermore subject to intermittences and recurrences.

The diagnosis of hysterical brachial monoplegia is generally easy. Of course, one can understand that a limited lesion of the internal capsule may determine a monoplegia with anæsthesia, but it is difficult to imagine such an affection without at least slight apoplectic symptoms, and the paralysis would be rapidly followed by contracture. An hysterical monoplegia with trophic troubles and dissoci-

ation of the anæsthesia might simulate a syringomyelia and the differentiation would be difficult without a knowledge of the previous history. The radicular paralyses of the brachial plexus are generally to be recognized by their distribution and by the characteristics of neuritic paralyses. Certain facts recorded by Brissaud and Lamy seem to indicate that hysterical subjects have a special tendency to anatomically systematized paralyses resulting from local irritation; a previous intoxication, by determining a local weakness, may have the same effect; lead poisoning, for example, may predispose an hysterical subject to a particular form of radial paralysis.

Crural monoplegias, which are more rare than those which we have just been considering, correspond in general to the description above given. We find the same segmentary anæsthesia under the form of a boot, a sock, or one leg of a pair of drawers. In the case of an alcoholic hysteric who had had a hemiparaplegia with incomplete anæsthesia following a fall on the left hip, the extensors of the foot were affected in a very markedly predominating manner. Adamkiewicz has reported a case in which there was hemiparaplegia with crossed anæsthesia, simulating Brown-Séquard's paralysis, but the anæsthesia stopped at the root of the limb.

The monoplegic contractures of the lower extremities occur under the same conditions as the paralyses. Contrary to what occurs with the latter, however, monoplegic contractures appear to be more frequent in the lower extremities.

Contracture of the upper extremity affects either flexion or extension, the arm in both cases being applied to the body. In the flexion type the forearm is flexed on the arm, the hand on the forearm. In the extension type the upper extremity is extended along the body, the position of the wrist varying in different cases. The hand is usually closed, but sometimes the fingers are contractured in the position of holding a pen. When the fingers are contractured in extension, they are at the same time approximated to, and firmly pressed against each other, and rarely are separated. In the closed-fist attitude, the thumb is sometimes covered by the other fingers, sometimes applied alongside the index, and sometimes carried farther forwards so that its terminal phalanx is in contact with the second phalanx of the middle finger (Richer). The different segments of the upper extremity may be contractured separately.

Contracture of the lower extremity is commonly a sequel of an attack, but traumatism such as sprains and the like is also a frequent cause. The various segments of the limb may be contractured independently, and in that case it is the foot which is more often affected, presenting the different deformities of clubfoot, more fre-

quently that of equinovarus with torsion inwards and very pronounced flexion of the toes. Exceptionally the toes are not flexed, more rarely we find pure equinus, and talus is still more rare. When the other segments of the lower extremity are affected, we find them usually in extension; but flexion may be observed, the thigh being flexed on the pelvis, and the leg on the thigh, so that the heel touches the buttock. In case of arthralgia of the hip the contracture is always one of extension.

In a general way the monoplegic contractures are accompanied by the same disturbances of sensibility as the corresponding paralyses; we usually find anæsthesia, but there are certain cases of painful contractures which merit a special consideration from a diagnostic point of view.

The motor troubles of hysterical patients do not present themselves under the hemiplegic or monoplegic form only, for paraplegias are not rare. These paraplegias may affect both the lower limbs or all four extremities; they are flaccid or rigid, complete or incomplete.

Paralysis of the lower extremities is one of the most frequent of all hysterical paralysis. It may be provoked by any of the ordinary causes, but the emotions play a specially important rôle in the etiology; fatigue is also a frequent cause, and I have seen a young girl whose paraplegia appeared after a dream of long continuance.

When the paraplegia is complete the patient is confined to the bed, and sensibility in the affected extremities is entirely abolished and even the sensation of displacement is lost; in consequence of this we ought to watch the position of the limbs in order that they may not contract vicious attitudes which might finally become fixed, by fibrotendinous contractions. We should note that the disturbances of sensibility are not always confined to the same regions as those of motility; we may see the anæsthesia arrested just above the knee or mount up to the umbilicus. Although as a rule troubles of sensibility are more frequent and more marked than those of motility in hysteria, we may see hysterical paraplegias in which there is no disturbance of sensation. Whether the paraplegia be flaccid or rigid it may be accompanied by vesical and rectal troubles, retention being the more common, although we may also observe incontinence. Trophic disturbances are rare; nevertheless we occasionally see marked atrophy of the muscles or even sacral bedsores. In spite of these accidents, however, the paraplegia may be recovered from even after it has existed for years.

In flaccid paraplegia the tendon reflexes are normal, and when they are exaggerated and we notice a tendency to epileptoid tremor, contracture is threatening. Spasmodic paraplegia is not exceptional

in hysteria. As a rule the limbs are in a position of extension with more or less marked adduction; the foot may preserve its normal position, forming a right angle with the leg, or it is in a position of equinus, in which case, the more frequent one, walking is impossible. The affection may be complicated with fibrotendinous retractions which remain after the paraplegia has ceased and mask the subsidence of the latter. We sometimes observe retention of urine in spastic paraplegia. Recovery from paraplegia with contracture may take place even after years, nineteen years in one case (Zeni).

The different forms of paraplegia, varying in intensity from paresis to paralysis with complete relaxation or inflexible rigidity, may simulate most of the paralyses following medullary lesions, Pott's disease, compression by syphilitic, tuberculous, or other lesions, multiple sclerosis, etc. The first step in the diagnosis consists in a search for permanent stigmata, but even when they are found we have not arrived at a certainty; for it is not rare indeed to see hysteria coincide with an organic affection of the nervous system, preceding or following it. Souques has made a careful study of the differential diagnosis of hysterical paraplegia with stigmata, and of organic paraplegia, embodying the results of his study in a thesis to which we are indebted for the following *résumé.*"

In organic paraplegia subjective sensations, dysæsthesia, formication, etc., are the rule; but in hysterical paraplegia, which is ordinarily accompanied by circumscribed anæsthesia, these subjective symptoms are rare. In spinal paralyses the electrical reactions are quickly changed, but are only exceptionally affected in hysterical paralysis. Trophic disturbances are rare in hysteria, although we may occasionally observe muscular atrophy; but decubitus with grave general symptoms is rare. Although in hysteria we may observe disorders of micturition, they are exceptional and are not accompanied by the purulent cystitis which belongs to the complications of organic paralyses.

Although, as we have just seen, the subjective phenomena are rare in hysterical paraplegia they are, nevertheless, occasionally seen, and twenty years ago Webb raised the question of an hysterical pseudotabes. Hysterical pseudotabes is the result of various conditions which may provoke hysteria, among them especially the emotions. The beginning of the trouble is usually slow and insidious, and its progress is gradual and often interrupted by periods of temporary improvement or of apparent cure. It is characterized chiefly by the inability to stand steadily and by an ataxic gait which is increased in the dark or when the eyes are closed. To these troubles are added sensations of numbness, of formication, of prick-

ing, lightning pains, a painful spinal point, gastric or anal crises, and vesical troubles. The resemblance would be complete were it not that the tendon reflexes are usually intact and the tabetic disturbances of vision, such as the Argyll-Robertson sign and pupillary atrophy, are absent. The differential diagnosis, however, must often be reserved and the nature of the trouble becomes manifested only by its further progress.

Generalized paralysis or contracture of all four extremities is rare in hysteria and is hardly ever seen except in individuals whose neurotic condition is inveterate. It is provoked by the same banal causes as other hysterical affections and is subject to oscillations and to recurrences. During some period at least of their evolution these paraplegias are usually accompanied by paralysis of various organs, of the muscles of the tongue, pharynx, thorax, or bladder, and there are usually also disturbances of sensibility and sometimes vasomotor troubles. The onset of the paralysis may be sudden, following an attack, a traumatism, or some mental shock; more often, however, the affection begins gradually and is slowly progressive. It begins usually in one of the lower extremities and extends first to the upper extremity of the same side and then invades the opposite side; it is the left side which is first affected as a rule and in which the paralysis is most pronounced. Flaccid paralysis often precedes the contracture. In the diagnosis of this affection we may exclude cerebral diplegias, in which the disorders of sensibility are wanting and which are accompanied by a special mental condition and by secondary contractures with the reactions belonging to them; cervical pachymeningitis and Pott's disease of the spine are accompanied by characteristic local symptoms. In spinal paralysis there is a loss of reflexes and a more general and more marked muscular atrophy than is seen in hysterical paralysis; furthermore the disorders of sensibility are wanting. Peripheral polyneuritis is accompanied by electrical reactions which are sufficiently characteristic apart from the etiological conditions. We must always remember, however, that hysteria may coincide with a gross organic lesion.

Tetany.—Under the name of intermittent tetanus Dauce has reported an affection which L. Corvisart described under the name of contracture of the extremities, or tetany, which latter term has prevailed since it was adopted by Trousseau. The patient has a sensation of formication in his hands and feet, afterwards a hesitation, and then an actual impediment in the movements of the fingers and toes. Soon the extremities become stiffened and the patient, although still capable of certain movements, can no longer contend against the contraction, which increases and becomes painful like a cramp. The

fingers are strongly pressed one against the other in the form of the "accoucheur's hand;" they then become flexed and the whole hand becomes flexed on the wrist. The toes, closely pressed together with the great toe beneath the others, become flexed on the sole of the foot and the latter becomes hollowed while the dorsum of the foot is arched and the heel is raised. The upper extremity may be flexed or extended but the lower is usually extended. In severe cases the contracture invades the muscles of the trunk and of the larynx, provoking suffocative troubles.

Tetany occurs in paroxysms which may be provoked by compression of the vessels, nerves, or muscles. This circumstance might have sufficed to direct attention to the hysterical nature of these troubles which have been noted by Raymond.[9]

We have already remarked that the existence of systematic paralyses in hysterical subjects had not been proven by unequivocal evidence and for us astasia-abasia, as a systematic paralysis, does not exist. The expression employed by Charcot and Richer in 1883, namely, "a motor weakness of the lower extremities through defective coördination in standing and walking," although more vague appears to me to be preferable; it records the fact of diminished energy coincident with a defect in the precision of movements. In this pathological state as in the physiological conditions there is a correlation between the energy, the rapidity, and the precision of the movements. As long as it has not been shown by methods of precision that the disturbances of coördination do not coincide with a defect in the other qualities of movement, we must regard astasia-abasia as an incomplete paralysis. We cannot conclude from the fact that a patient can stand in water but not on land that he has a systematic paralysis, we can only say that he is incapable of holding himself erect when he is not relieved of a part of the weight of his body equal to that of the volume of water which is displaced. A patient with astasia-abasia is in an analogous position; he is incapable of standing or of walking when he has his weight to support, but he can do so when relieved of this weight. In healthy persons a diminished force of movement, under the influence of fatigue, loss of blood, etc., necessarily brings with it a diminished precision of movement, a certain incoördination which is exaggerated in hysterical patients by reason of their tendency to spasm.

Whether or not they deserve the name of astasia-abasia, incomplete paraplegias, with or without incoördination, are none the less worthy of special mention. The characters which have given to them this special designation are the impossibility of walking (abasia) and that of standing (astasia), the first never passing into the second,

contrasting with the possibility of executing all the movements concerned in walking while the patient is lying down. This contrast is only apparent, as I have already remarked, for when we measure the movements made while the patient is lying down we see that they are weakened and retarded.

Astasia-abasia may occur under the paretic or paralytic form, it is the loss of power which characterizes it; Charcot compared such a patient to a very young child who had never yet tried to walk, making his first step while held by the nurse. This form has different degrees; some patients can walk when they enlarge their base of support by separating the legs. When the paresis is more marked on one side the patient can walk only by putting forward the same foot, bringing the other up to it. This form may present rapid oscillations of intensity, the patient being able to make a few steps and then suddenly falling. The abasia may also occur in paroxysms (Ladame).

Motor incoördination is often added to paralysis. There exists a choreiform astasia-abasia, or flexion type of Charcot, in which standing is disturbed by sudden flexion of the pelvis on the thighs and of the thighs on the legs. These troubles are increased during walking; at each step the patient bows and straightens himself up alternately by movements which become more and more violent and rapid as he goes on. He threatens to fall at each step and seems to be struggling to maintain his equilibrium.

In the tremulous astasia-abasia of Charcot, walking is disturbed by a trepidation which is an exaggeration of that which we sometimes see in spastic paraplegia when attempt is made to stand the patient on the extremity of his rigid feet. The trepidation may increase to such an extent as to become a sort of stamping movement or even true leaping—a saltatory spasm (Brissaud).

These forms of paralysis or of spasm are much more tenacious in adults than in children. Their nature may be rather difficult to distinguish, for, as Charcot has observed, these motor troubles may be the only hysterical manifestations. When there are no stigmata we must search for the symptoms which may be associated with incoordination in cerebellar lesions and in multiple sclerosis. The saltatory form may be produced by transverse myelitis, compression of the cord, lateral sclerosis, or perhaps even by locomotor ataxia.

In neurasthenia and allied states there have been described anxious fears of the execution of certain acts, among others the fear of standing alone (atremia of Neftel, stasophobia of Bouveret, stasobasophobia of Debove and Boulloche). Binswanger has regarded these as emotional forms of astasia-abasia, but the anxiety of these phobias gives rise to special symptoms.

Disorders of Speech.

The troubles of speech which we shall now study might be regarded as motor aphasic troubles, but it seems to us more practical to consider them among the disturbances of phonation.

Hysterical *mutism* attracted hardly any attention before the work of Revilliod in 1883, but it has been specially studied by Charcot and Cartaz.[50] It is now distinguished from the two forms of aphonia and dysphonia.

Hysterical mutism is found almost as often in men as in women; it is more frequent in adults, but it has also been seen in children of five or six years. Ordinarily its onset is sudden, following an hysterical attack, a traumatism, or some powerful emotion. Sometimes it comes on gradually, following an affection of the throat or larynx. It may be preceded by a period of dysphonia or stammering. Hysterical mutism has been regarded by Charcot as a systematic impotence. "Although," he says, "the patient may have preserved the power of executing all the ordinary movements of the tongue and lips, and although he may move these organs perfectly in all directions so as to blow or whistle as in the normal state, it is impossible for him to articulate a word even in a low voice, a whisper in other words, or even to imitate the movements of articulation which he sees made before him, however much he may bend his attention to it. The patient is then dumb in the strictest acceptation of the word, in that he cannot utter a word. I will add that he is more than dumb, for while it is possible for a deaf-mute to utter even loud cries, the hysterical mute—note well this singular characteristic—is aphonic, often absolutely so, in that he cannot utter the least cry."

Those authors who, with Charcot, believe that there is motor integrity of the vocal organs have never verified this integrity by methods of precision; and others have often noted motor troubles, even those which are appreciable without a dynamometric or chronometric examination; thus in sixty per cent. there has been found either paralysis or contracture,[51] and weakness of the laryngeal muscles has been found as well as that of the tongue and lips. As a rule the hysterical mute hears and understands perfectly, makes efforts to speak, and not succeeding writes down what he cannot make understood. Sometimes, however, deafness coincides with mutism. Exceptionally agraphia complicates the mutism, and this may be merely transitory or persistent. It is not uncommon to see mutism associated with a certain degree of facial paralysis or spasm. The paralytic nature of mutism is shown by a circumstance in its course: just as we often see the loss of speech preceded by dysarthria or stammering, so we may

also see mutism give way to stammering before disappearing entirely. But frequently enough the mutism disappears as rapidly as it had come on, following an attack, an emotion, or a shock of some kind. In a case in which all the movements of the tongue and lips appeared normal, in which I had found a slight weakness, the energy of these same movements increased more than a third after the return of speech following an attack. Hysterical mutism may last a few hours or several years; its prognosis is therefore far from being always benign.

Stammering, which may precede or follow mutism, may also constitute an independent manifestation recurring under the same conditions as mutism; it is always accompanied by disturbances of motility of the tongue, such as paresis, spasmodic deviation, or tremor. Hysterical stammering occurs furthermore under various forms, and we observe also other dysarthrias, such as stuttering, lisping, and so forth, which manifest themselves under the same conditions and with the same paralytic or spastic accompaniments. There is sometimes observed a defect in articulation which consists in scanning the words, like the speech defect of patients affected with multiple sclerosis.

Respiratory Spasms.

The various spasms of articulation may be compared to the respiratory spasms with which they sometimes coincide. The respiratory spasms are usually rhythmical. Pitres divides them into three groups: 1. Simple respiratory spasms, the most frequent, in which convulsive twitchings occur exclusively during either inspiration or expiration; the expiratory spasms which cause phenomena more or less analogous to cough, or snorting; the inspiratory spasms which produce noises resembling hiccough, yawning, or sniffing. 2. Mixed respiratory spasms in which the movements of both inspiration and expiration are disturbed by the spasm. 3. Complicated respiratory spasms, in which the convulsive twitchings involve the muscles both of respiration and of phonation (cough, barking, mewing, howling, bellowing, etc.). The hysterical laryngeal noises are often inspired by the imitation of animals which have attracted the attention of the patient shortly before the onset of the spasm. The influence of contagion sometimes manifests itself in epidemics.

Hysterical *cough*, like other laryngeal noises, is sometimes continuous, at least during the waking hours; but more frequently it occurs in the form of paroxysms, sometimes preceded by phenomena of the aura and followed by a stage of convulsion or delirium. The laryngeal spasms may occur in consequence of a lesion of the air passages, but they are usually not accompanied by any grave disturbances ex-

cept fatigue; we seldom observe any effects of the dyspnœa or suffocation. These spasms are more frequent in girls and in young subjects in general; they are usually of rapid onset, following an emotional disturbance or an attack, and their termination occurs often in the same manner; yet not infrequently they may continue for months or years. The hysterical stigmata which often accompany them enable us to distinguish them from the analogous spasms occurring in the *maladie des tics* or in paramyoclonus multiplex. Cough, barking, aphonia, and sneezing may alternate in the same individual, and these spasms may also coincide with choreiform movements; chorea and hysteria approach each other closely. Cough, hiccough, sobbing, yawning, sneezing, laughing, etc., may receive special descriptions among the symptoms of hysteria, but all these spasms are of interest only in their general aspects and in respect to their accompaniments. Briquet has seen asthma alternating with an hysterical paralysis; Charcot and Weir Mitchell have seen a rather peculiar form of dyspnœa in hysterical subjects, which was characterized by a very superficial respiration repeated 170 to 180 times a minute, without any anxiety, pain, trace of cyanosis, noise in the chest, or acceleration of pulse. This hysterical increase in the respiratory movements occurs in paroxysms during the day, but ceases during sleep; it is announced by phenomena recalling those of the aura, and after a variable duration of several hours is terminated by a flood of tears like an ordinary attack. When these crises of tachypnœa appear alone, unaccompanied by any other hysterical manifestation, they may be mistaken, for a time at least, for dyspnœa of organic origin. Hysterical dyspnœa may be accompanied by bronchial secretion and sometimes by hæmoptysis. Recently a case has been reported in which there was a tracheal râle, more marked during expiration and comparable to the râle of the dying (Rist).

We also see in hysteria suffocative attacks with hæmoptysis, which are caused rather by a vasomotor trouble than by a vicarious congestion of the lungs; for although hæmoptysis may coincide with amenorrhœa, this coincidence is often wanting, and furthermore hysterical hæmoptysis may be encountered in man. It is often connected with the attacks, occurring at the end of one with a blowing respiration and a rattling sound in the trachea. The hemorrhage may be more or less abundant and may coincide with other losses of blood, such as epistaxis or hæmaturia. Hæmoptysis occurring apart from the attacks is usually seen as an isolated symptom. Hysterical pulmonary hemorrhage is accompanied often by superficial dysæsthesias of the chest, usually on the side on which the stigmata predominate. Auscultation enables us to recognize signs of congestion of more or

less intensity, and sometimes even pleural irritation. There is nothing peculiar in the character of the hemorrhage, which in general is not frothy as it is in the hæmoptysis of the tuberculous. Sometimes the hemorrhages recur at short intervals and are very abundant; at other times the blood is coughed up in small amount and at rather long intervals. It is not very uncommon to see neuropathic hæmoptysis accompanied by a certain acceleration of the pulse, a slight elevation of temperature, emaciation, and night sweats which simulate tuberculosis. This resemblance may be the stronger in that certain hysterical subjects present more or less constantly, but principally in the morning, a weakness of respiration more marked on the side on which the stigmata predominate.

This weakness of respiration, more marked on one side, appears to be due in great measure to a paretic condition of the thoracic muscles of inspiration; but paralysis of the diaphragm may also contribute to it. I have found that the movements of the diaphragm may be restricted on the paralyzed side in hysterical hemiplegics.

An isolated paralysis of the diaphragm, lasting for several months, has been observed by Briquet. This paralysis is manifested by a very short respiration, a muffling of the voice, and a shortness of breath, which is increased on the least movement. "During inspiration the diaphragm does not contract, but is pushed up into the thorax, and there is then a very pronounced depression at the base of the thoracic cone, and especially in the epigastric region; on the other hand, this part projects during expiration, which is the exact opposite of what takes place under normal conditions." Respiratory troubles, leading even to suffocation, might be produced under the influence of contracture of the diaphragm, the possibility of which in hysteria can hardly be doubted, but the existence of which has never yet actually been demonstrated.

The *larynx* may alone be affected in hysteria. The troubles of this organ consist either of disturbances of sensibility, such as anæsthesia and dysæsthesia, or of motility, such as paralysis, contracture, or spasm. The ordinary general exciting causes of hysteria may play a part in the production of these laryngeal affections, but local irritations and affections of the uterus, an organ which is evolutively related to the larynx, are especially active. Dysæsthesia may provoke a special emotional state (phonophobia of Coën), in which the patient is afraid to speak aloud, and contents himself with whispering, which spares the movements of the larynx. It is most often anæsthesia which accompanies the motor troubles, but in certain cases the spasms are seen in conjunction with dysæsthesia.

The study of the localization of paralyses of the larynx offers con-

siderable difficulty by reason of the complexity of these troubles. The paralysis is often bilateral, but it may be unilateral, and may be combined with a certain degree of contracture of the antagonists. These combinations produce veritable symptomatic paradoxes; a paralysis of the dilators of the larynx, which ought to produce aphonia, gives rise instead to an inspiratory hoarseness with dysphonia, while expiration is free and the voice is unaffected. Aphonia is, however, the functional trouble which is most frequently associated with paralyses of the larynx in hysteria.

Aphonia consists in an impossibility of speaking aloud, while whispering, which necessitates the movements of articulation only, is still possible. It is thus very distinct from mutism, with which, however, it may coincide, as I have recently had occasion to observe. Its onset is, in general, gradual when it follows an irritation of the larynx or throat, but rapid when it follows an hysterical attack or a shock. The respiration in both its phases remains free, but no sound whatever can be emitted. Some patients, nevertheless, speak in their dreams (Thaon, Gougenheim), or they may sing (Griffith). We may sometimes note the coincidence of an anæsthetic area on the skin of the anterior portion of the neck, and the laryngeal mucous membrane is also usually anæsthetic. A laryngoscopic examination will enable us to note the presence or absence of a local lesion, but very often we are left in doubt as to the seat of the paralysis or contracture. Hysterical aphonia is grave only under certain professional conditions, but it may constitute an annoying inconvenience because of its duration, which may be a question of months or years. On the other hand, it may last only a few hours or during the interval between two attacks. Even when it comes on gradually and is provoked by a local lesion, it may disappear suddenly after an attack or in consequence of some emotional disturbance. It may return also under the same conditions.

Spasm of the adductors of the vocal cords is shown by a more or less intense inspiratory dyspnœa and hoarseness. It may give rise to symptoms of very threatening suffocation, for the relief of which tracheotomy may appear justifiable.

Digestive Disturbances.

Among the preludes of hysteria in young girls, Briquet has noted gastralgia as almost constant, and the troubles of digestion in general are certainly very frequent.

Gustatory anæsthesias and dysæsthesias appear to play an important part in the pathogenesis of hysterical perversions of appetite, such as pica, malacia, etc., or repugnance to certain articles of food.

In rare instances we may see a dysæsthesia which renders all contact of liquid or solid food painful (Fabre). Sometimes these dysæsthesias are accompanied by ptyalism, which may also exist alone.

Mastication and deglutition may be impeded by spasm of the masseters or by glossolabial spasm, but an impediment to the ingestion of food is offered more frequently by *spasms of the pharynx or œsophagus.* Spasm of the pharynx forms a part of the first period of the attack, and it has always been regarded as playing an important part in the origin of the globus hystericus. It is usually intense enough to prevent any attempt at deglutition. A great many hysterics, however, have a spasm under the form of a prodrome for some hours before an attack, and sometimes this forms the only manifestation of the attack. This isolated spasm of the pharynx occurs ordinarily under the remittent tonic form, but Bouveret has described, under the name of hysterical *aërophagia*, a clonic spasm of the pharynx, having for its cause an excessive hyperæsthesia of the muscles of the pharynx. The spasmodic movements follow each other rapidly, under the form of paroxysms, which last for two or three minutes, and are succeeded by a period of calm. Each movement is accompanied by a sonorous sound, and on auscultation of the œsophagus and stomach a gurgling noise is heard. From time to time there is an eructation of pure air. Involuntary deglutition of air occurs rather frequently, under the form of spasmodic movements, and it appears to be rather frequently associated with the production of inodorous eructations, tympanites, and borborygmi. The results of this swallowing of air are made clear by the suppression of the eructations and borborygmi when the patient keeps his mouth firmly closed.[22]

Spasm of the pharynx and œsophagus occurs rather frequently, under the form of more or less permanent contracture—œsophagismus (Mondière). This spasm may be seen in children, but it is especially frequent in adults, and particularly in women. Like all permanent spasms, it may be produced slowly, in consequence of a local irritation or of one in the neighborhood, or suddenly following an attack or emotional excitement. Sometimes there is a remittent stage before the spasm becomes permanent. It may occasion regurgitations, œsophageal vomiting, which may be mistaken for gastric vomiting when the spasm is located at the lower part of the tube; when it is located in the upper part, the patient spits out rather than vomits the food which cannot pass the obstruction. When by a great effort the bolus is passed beyond the obstruction, it is often actually vomited, which shows that the spasmodic condition is not confined to the œsophagus alone. Sometimes the passage of food by the strictured point occasions the explosion of an hysterical attack. Sometimes

certain kinds of food can pass the obstruction, while others are arrested; this is rather characteristic of hysterical dysphagia. The passage of a sound may give us valuable information concerning the nature of the evil, showing that no organic lesion exists, or sometimes provoking an hysterical explosion, in consequence of irritation of an hysterogenic zone.

According as the spasm is continuous or remittent, according as it permits the passage of a greater or less quantity of alimentary substances, it exercises a variable influence upon nutrition. Death may result; but when the spasm suddenly relaxes under the influence of emotional causes or of an attack, we see the patient's flesh return with astonishing rapidity.

Spasm of the stomach often coexists with that of the œsophagus; it is brought into evidence when food which has passed the œsophageal obstruction is almost immediately vomited. But gastric spasm may exist alone, being provoked by dysæsthesia of the mucous membrane of the stomach, which is intolerant of the least contact. It is to this spasm that are due the hysterical vomitings, which are characterized by their frequent repetition and continuousness. Spasm of the pyloric region appears to be active in the production of the cramps of the stomach, which are so common in hysterical patients. This vomiting may be observed at all ages, but it is more common in adolescence and adult life, and especially in women. Continuous, incoercible vomiting may come on suddenly at the conclusion of an attack, or it may at first be intermittent. It occurs only after the introduction of food, is always alimentary, and follows ingestion almost immediately; it is almost invariably painless. It is sometimes complete or nearly so, nearly all the food being rejected, and emaciation is then rapid. At other times there is a selection of the rejected matters, some articles being retained; or, again, the vomiting is incomplete without selection, not all the ingested food being vomited, and then the emaciation is less rapid. But as in any case we have to do with a remarkably obstinate trouble which may last for months, marasmus finally sets in, and death is the outcome. The instantaneousness and the painlessness of the vomiting may cause it to simulate that due to organic cerebral lesions. Sometimes, after a period of obstinate vomiting, we see the production of dilatation of the stomach, with all the troubles which are associated with this condition. This dilatation might be compared, as regards its mode of production, to the paralyses by exhaustion which we sometimes see in hysterical subjects. It is not improbable that these paralyses of the muscular wall of the stomach may occasionally be primary and play an important rôle in the pathogenesis of the anorexias.

Gastric spasms do not always occur without pain, and, indeed, they are often associated with the gastralgias which are so frequent in women, especially in young women and in those approaching the menopause. Sometimes contact of food with the mucous membrane of the stomach provokes not only vomiting, but even severe convulsive attacks. The existence of hysterogenic zones in the mucous membrane of the stomach has been assumed by some authors (Sollier, Gilles de la Tourette).

Often, also, *gastralgia* is manifested without any spasmodic accompaniments and without vomiting. It sometimes occurs at other times than during a meal, being excited by emotions, but more commonly it follows immediately the ingestion of food. It consists in a sensation of burning, tearing, or crushing, which comes on suddenly, explosively as it were, and which reminds us of the gastric crises of locomotor ataxia. These gastralgias, especially when they are persistent, are accompanied by throbbing in the temples, ringing in the ears, palpitation, a feeling of suffocation, ovarian and spinal pains, and, in a word, most of the painful phenomena of an attack of hystero-epilepsy. They may last for several hours, even when there is no gastric cause for them; indeed, the attempts at vomiting when the stomach is empty are especially painful. When these crises are frequently repeated, a progressive emaciation results, and often, notwithstanding the preservation of the appetite, the patients refuse absolutely all food, in consequence of which gradual inanition results. Sometimes the voluntary abstention from food gives rise, as in other conditions, to true anorexia. The gatralgic crises are therefore grave, not only because they are extremely painful, but because they may cause death by marasmus (Briquet).

In connection with these gastric troubles we may mention the paræsthesia, manifested by a tickling or a sensation as of something crawling in the stomach, which is complained of by some patients.[53]

Hæmatemesis.—The gastralgia is sometimes seen in connection with vomiting of blood, but the latter often occurs alone. It not uncommonly appears at the period of the menstrual flow, of which it may modify the amount. Sometimes it coincides with the suppression of the menstrual flow, but it has no necessary relation with the latter, and it may occur in man. The vomiting of blood, seen in connection with other hysterical symptoms, appears in consequence of a blow on the epigastric region, or of some strong emotion, or in relation to an hysterical attack, which it may precede, accompany, or follow.

As a rule, the hæmatemesis is preceded by a pain, with a swelling of the epigastric region. This pain radiates towards the ovaries or

the back, and is accompanied by precordial anguish, pharyngeal constriction, and other variable phenomena of the hysterical aura; then the vomiting occurs, usually without effort, in one or two attacks. The patient often faints. The vomiting may occur but once or may be repeated in a series. The blood vomited is sometimes pure, sometimes mixed with food particles; its color varies greatly, but as it is usually vomited immediately after its extravasation, it is rarely clotted or black, as in melæna. The blood is often diluted with a watery fluid, which may be saliva or a product of gastric secretion. These attacks of vomiting of diluted blood were recognized by Lasègue, but were studied especially by Josserand, Mathieu, and Milian, who gave them the name of *pituites hémorrhagiques des hystériques*, because of their frequent occurrence in the morning.[54] The amount of blood which is vomited varies considerably; when it is abundant a more or less lasting anæmia may result. Sometimes hysterical hæmatemesis recurs with a certain periodicity. The diagnosis is easily made. A direct examination will enable us to exclude a buccal hemorrhage; hæmoptysis is accompanied by special symptoms; but it will be difficult to eliminate ulceration of the stomach, for Gilles de la Tourette has shown that round ulcer is frequent in hysterical subjects, and the exquisite cutaneous hyperæsthesia in the epigastric region, which he notes in the case of ulcer, may perhaps occur in hemorrhage from vasomotor troubles. In any case, this frequent occurrence of round ulcer in hysterical subjects is interesting to note, for it may lead us to fear death as a consequence of gastric troubles which we are more accustomed to regard as associated with no gross lesion. The pain which sometimes accompanies gangrene of the skin would suggest the idea that ulcer of the stomach may be the result of an analogous process.[55]

Anorexia may be associated with gastralgia and incoercible vomiting, but it may also be a primary condition. Under the name of hysterical anorexia, Lasègue has described a special condition, rather frequently encountered in girls from fifteen to twenty years of age, suffering from more or less definite hysterical troubles. This consists in the more or less absolute refusal of food, while there is found no digestive trouble to which it could be justly attributed. The loss of taste and of sensibility in the digestive apparatus, which are necessarily accompanied by disturbances of secretion, may have a part in its production. The patient complains first of a malaise, of a sensation of fulness or of distress in the stomach, which leads her to diminish the amount of food taken; then, instead of recognizing that inanition has aggravated the trouble, she expresses repugnance to food, and finally refuses flatly to take any nourishment. The disease

follows then a fatal course, in which we may recognize four rather distinct stages: 1. At the beginning of the trouble, when the patient complains of gastric pain, cramps, or sensation of fainting, pallor, sweats, etc., but when there is no nausea or vomiting, we may question whether we have not to do with a gastralgia, the cause of which remains to be discovered. 2. Then the mental condition becomes aggravated, and after some hesitation the patient seems to have become convinced that there is no hope of relief except in abstention from all food, and she then diminishes gradually the amount taken. The special nervous condition of these patients gives them a peculiar power to tolerate abstinence, and for weeks and months they do not appear to suffer from being deprived of nourishment. They emaciate but little, are in good spirits and lively, and have a constant need of being on the go; indeed, they do not appear to suffer in the least. The result of this absence of general troubles is that the physician is absolutely without authority, in so far as that, although the patient may be willing to take the medicines prescribed, she cannot be turned from her determination to take no food, especially as those about her can hardly be made to believe that the condition is a grave one. 3. But still the anorexia does not yield. Those about the patient beg and entreat her to take food, and the delicacies of the table are multiplied in the hope of tempting her. "The excess of insistance arouses an excess of resistance;" we may demand, but the patient intrenches herself behind the absence of all suffering. She is as strong and as lively as ever; she maintains a peculiar quietude, a sort of pathological contentment; and she opposes to all an invincible force of inertia. 4. Little by little this tolerance wears away, the organism becomes exhausted, emaciation becomes more and more apparent and makes rapid progress, the abdominal wall retracts, the skin becomes dry, and the patient grows weak and can no longer stand without vertigo and faintness. Then the quietude of the relatives becomes transformed into a veritable passion of anxiety. Little has been added to the clinical picture traced by Lasègue, in the *Archives générales de Médecine*, in 1873. In 1874 Gull recalled the fact that he had recognized this condition in 1868, which he had described under the name of nervous apepsia. He relates a certain number of interesting facts, and especially the possibility of the existence of this peculiar anorexia unassociated with any other hysterical trouble. The affection may be seen in men also. He proposed the name of nervous anorexia, which term was accepted by Charcot. He insisted, furthermore, on a weakened state of the respiratory and circulatory functions, and reports a case in which the pulse fell to 50 and the respirations to 12 in the minute. Gull mentions also the possibility

of a certain lowering of temperature, and he deduces from this a therapeutical indication. It follows, indeed, from the experiments of Chossat that animals in a condition of extreme emaciation can no longer digest without the help of external warmth. Gull proposed to warm the vertebral region with a large rubber tube filled with hot water. Dowse recorded, instead of a slowing, an acceleration of the pulse, which beat 120 times to the minute; and he described the change taking place under the influence of treatment—a patient so emaciated as to present a senile appearance, regained a physiognomy in keeping with her age within a few weeks.

The treatment of this anorexia consists in forced feeding and isolation, for the relatives and friends are the very worst nurses in these conditions. Charcot has especially insisted upon the value of isolation in the treatment of these patients. Even when the anorexia is accompanied by nausea and vomiting the forced feeding may be useful, for food introduced through the tube may be retained when that which is swallowed may be immediately rejected. Relapses are not rare, and each new attack is, in general, more grave than the preceding, if we do not at the very first symptoms at once have recourse to isolation. Death may take place from syncope, from the excessive weakness, or from tuberculosis, the onset of which is favored by the malnutrition.

Lasègue had noted the mental perversion which often coincides with anorexia. At the beginning we often observe a more or less marked agitation, frequently persisting to an advanced stage of the condition, manifested by an incessant need to walk and to exercise to a most fatiguing degree. Many patients compress themselves to an excessive degree, not only with the corset, but often with bandages and with belts placed immediately on the skin. Many are delighted to be thin, and attribute a coquettish attractiveness to their leanness, even when it has progressed so far that the skin of the hands, cold and clammy as a frog, has lost its elasticity and remains in folds where it has been pinched up, and when the face has assumed an earthy hue and is seamed with wrinkles. It has even been asserted that these patients are affected with illusions, especially visual ones, such as macropsia (Sollier), under the influence of which the food that is presented to them seems to be too great in amount. But when a child heaps his plate too full, it is not because of a visual defect, a micropsia; it is because he thinks only of his appetite. The hysterical patient does exactly the same, but in an inverse sense.

From the existence of these psychic troubles and of affectional disorders, characterized especially by the abolition of all altruistic sentiments, and from the possibility of its sudden origin and of its

cure as the result of some emotion, we might conclude that hysterical anorexia is at bottom a mental trouble. But the rather frequent coincidence of dilatation of the stomach—that is to say, of muscular paresis and of disorders of sensibility, such as anæsthesia and dysæsthesia, the relation of which to anorexia could hardly preëxist in the mind of the patient—forbids us to deny the existence of any previous organic lesion. Emotion includes somatic phenomena, which are very capable of rapidly modifying the gastric functions, and the sudden cures which we often see as the result of isolation do not prove that the trouble was primarily psychical.

Certain facts would appear to be of a nature showing that in hysterical anorexia there are quantitative and qualitative modifications of the gastric secretions, which would justify the term, primarily adopted by Gull, of apepsia. But observations on this point are still too contradictory to enable us to arrive at any positive conclusion.

For a long while it was thought that hysteria gave to those suffering from anorexia the privilege of living, and even of not emaciating, without eating. But those who do not emaciate take a certain amount of food and do not reject by vomiting all that they have swallowed: and those who do not take or retain any food emaciate and die. Ch. Richet has recently shown that, in hysterical anorexia, the alimentation may be reduced to 12 or 9 calories per kilogram in the twenty-four hours, instead of 40 to 55; the respiratory quotient to 0.21 to 0.49, instead of 0.69 to 0.92.

The *intestine*, like the stomach, may be affected in hysteria. We have already referred to the existence of spasms which may be revealed by borborygmi. But disturbances of motility of the intestine manifest themselves most frequently under the form of paralysis, which occasions a more or less generalized meteorism. This meteorism or hysterical tympanites is often accompanied by enteralgic pains or colic. The painful phenomena which accompany tympanites and which give rise sometimes to symptoms of general reaction may simulate peritonitis, and have therefore received the name of peritonism (Gubler).

General meteorism may arise very suddenly, either during the attack or after it, or as a result of the ordinary exciting causes of hysterical phenomena; at other times its progress is slow. The abdomen is in general uniformly enlarged, but the tension of the recti muscles may give it a bilobate form. The skin is often exceedingly sensitive, while deep pressure is better borne. Percussion usually gives a tympanitic sound, except when the distention is very great, in which case we may obtain absolute flatness, just as in cases of tympanites of other nature. When the distention is considerable,

there result mechanical disturbances of the pulmonary and cardiac functions, which may threaten asphyxia. The rapidity of the appearance of meteorism and its equally rapid disappearance under certain circumstances may be explained, in part at least, by a paralysis of the muscles of the intestine, which do not resist the pressure of the gas, while the sphincters do resist it. The reduction of meteorism under the influence of chloroform anæsthesia might be explained by a gradual relaxation of the sphincters, allowing the gases to escape insensibly. An attempt has been made to explain the meteorism by an instantaneous production of gas. This is not at all proven by the fact that Brodie was able to abstract gas by the rectal sound and noted that the patient floated in the bath; but neither can it be denied because of the absence of an audible elimination at the moment of the disappearance of the swelling. The insensible elimination of the gases of the intestine is a physiological fact, and is effected by relaxation of the sphincters under the influence of some emotion or of chloroform anæsthesia. Furthermore, the ordinary coincidence of constipation and of retention of urine with the meteorism may show that the contracture of the sphincters plays a part in offering an obstacle to this insensible elimination.

Hysterical tympanites may be of very short duration, especially when it is related to convulsive or emotional attacks; but it may also last weeks or months. It disappears rapidly, as a rule; but when it has occasioned a very great enlargement of the abdomen, like all permanent distentions, it may cause cracks which leave indelible cicatrices (Ebstein).

Sometimes the pain assumes an acute character, and if the meteorism is accompanied by dyspnœic phenomena and especially by acceleration of the pulse, and if there is also vomiting with an altered expression, we may be led to fear that we have to do with an acute peritonitis. A differential diagnosis could then be based only upon a knowledge of preëxisting hysterical symptoms, and especially would this be our only resource if nervous fever were also present. The latter, however, is of very rare occurrence. Spencer Wells has shown that in hysterical meteorism the administration of chloroform may instantaneously relieve the distention of the abdomen; but this is a diagnostic measure which might not be wholly innocuous if the case were one of acute peritonitis. Sometimes hysterical meteorism resembles tuberculous peritonitis, and, in the absence of stigmata, might easily be taken for it, especially as tuberculous peritonitis frequently exists without thoracic troubles. When the distention is extreme and the percussion note is dull, meteorism may be mistaken for a cystic tumor of the ovary; and Spencer Wells gives no absolutely dis-

tinctive sign, except the disappearance of the tympanites under the influence of chloroform. The resemblance to these phantom tumors may be especially striking in cases of localized pneumatosis. The pathological physiology of localized meteorism is obscure; but we can hardly understand how this condition could be the result of spasm of the muscles of the abdominal wall, and we therefore arrive by exclusion at a theory of localized contractures of the intestinal muscles. The existence of these localized contractures is the more probable, since, coincident with meteorism in hysterical subjects, phenomena of internal strangulation with stercoraceous and bloody vomiting have been observed. But stercoraceous vomiting may occur without obstruction, in consequence of a reversed peristaltic action of the intestine, as has been shown by the fact that fluid introduced by enema has been shortly after passed by the mouth.

Phantom tumors may be located in any region of the abdomen, although more frequently they simulate tumors of the uterine adnexa. I have observed a case in which there was a swelling limited to the right hypochondrium, with cutaneous dysæsthesia and more deeply seated shooting pains, suggesting an hepatic colic symptomatic of a tumor. But it would appear that there are facts which point to the existence of an hysterical hepatalgia, and there is really no good reason for refusing to admit the existence of hepatic colic with icterus in hysterical subjects when we know how great an influence the emotions have upon these pathological phenomena.

Under certain circumstances meteorism coinciding with suppression of the menses and the presence of vomiting may suggest pregnancy. When this idea is accepted by the patient, the early symptoms are soon accompanied by other phenomena, such as a secretion of milk and a peculiar mental condition, which may continue even after the limits of a normal pregnancy are exceeded. At the time of the normal termination of pregnancy we may observe pains which would appear to announce approaching labor.

Intestinal amyosthenia may cause obstinate constipation. At other times we may observe alternating constipation and diarrhœa resulting from an intestinal hypersecretion.

The anal sphincter may exceptionally be the seat of contractures or paralyses. Paralysis of this part with anæsthesia giving rise to involuntary evacuations would appear to be exceedingly rare. Spasm, which is less rare and which often coincides with urethral spasm, is ordinarily associated with dysæsthesia of the integuments. Local irritations, such as fissures, are especially apt to provoke it.

We have yet to mention among the intestinal troubles hysterical diarrhœa. This may come on suddenly, induced by emotional causes,

but may also set in without any apparent cause. Its duration is indefinite. It occurs ordinarily in crises, and alternates with other troubles.

Disorders of the Urinary Organs.

The disturbances of the urinary secretion have already been studied in the section on the trophic disorders of hysteria, but the urinary apparatus may be the seat of other troubles in the hysterical. Among the disturbances of sensation may be mentioned renal neuralgia, which may be mistaken for nephritic colic, with radiating pains and hæmaturia. The possibility of this hæmaturia, which occurs under the form of more or less prolonged paroxysms, was admitted by Sydenham, Sauvages, Boyer, and Chopart; and the recent observations of Morris, Sabatier, and others would appear to confirm the belief of the earlier writers. Hæmaturia may occur apart from the nephritic crises; the painful paroxysms excited by floating kidney are favored by an hysterical soil. Finally, wandering kidney is regarded by Albarran, justly in my opinion, as a stigma of degeneration. It sometimes coincides with other anomalies.

The existence of an hysterical *polyuria*, independent of attacks and more or less permanent in character, has been demonstrated by Kien and Kuhner, and especially by Lancereaux, who have shown that this manifestation of hysteria is frequently connected with alcoholism, a relation which has also been demonstrated by other observers. The onset of hysterical polyuria may be rapid or gradual; the affection often follows some excess, any emotional storm, or a traumatism. The amount of urine excreted and the frequency of micturition are extremely variable; fifteen or even twenty-five litres may be passed in the twenty-four hours (Mathieu, Hirtz and Fraenkel). The abundant secretion and frequent micturition lead to insomnia and a peculiar neuropathic state, so that a psychical pollakiuria is soon added to the mechanical pollakiuria. The only chemical characteristic would be an increase in the chloride of sodium; the phosphates are usually of normal proportion, the urea varies with the diet, and there is neither sugar nor albumin. The urine is clear and limpid. Sometimes, when the polyuria is the only symptom, it may be diminished under the influence of suggestion.

As a rule, the polyuria is accompanied by polydipsia, and the thirst is in itself sometimes exceedingly distressing. But, although the mouth is rather dry, the tongue as a rule preserves its normal color; we do not see the blackish coating of the tongue or the dental caries so frequent in diabetic patients. The thirst, when it is very intense, tends to increase the insomnia and the condition of prostra-

tion which is often its consequence. The sufferers from polyuria may take alcohol in considerable amounts, and also active medicaments in large dose, because of the rapid elimination. In spite of associated polyphagia the emaciation is marked. The arterial tension increases as a result of the polydipsia.

This polyuria is very obstinate and is subject to recurrences; it may disappear and then return under the influence of trivial causes. It is of more frequent occurrence in man, and in its gravity resembles the other manifestations of masculine hysteria. The prognosis is aggravated often in consequence of the hypochondriacal preoccupations which accompany it. The diagnosis may be based upon the history of the case and upon the neuropathic accompaniments, upon the mode of onset and the remissions, upon the absence of the signs of diabetes mellitus, upon the possibility of cessation under the influence of hypnotic suggestion (Babinski) or suggestion in the waking state, or upon the effect of medicines capable of producing an illusion when they are given with authority and conviction.

The reality of hysterical *ischuria* or *anuria* has long been doubted because the condition often coincides with vomiting, which has led writers to regard the phenomena as a vicarious excretion of urine; and because, on the other hand, in several cases, and especially in the well-known case of Nysten, it has been possible to detect simulation. The cases of Laycock, however, are worthy of credence; but it is especially to Charcot that hysterical anuria owes its right of citizenship in pathology. A patient whom he had watched for several years at the Salpêtrière served to demonstrate, under favorable conditions for observation, the reality of ischuria, the coincidence of vomiting, the abundance of which varied inversely with the amount of urine excreted, and the existence of a certain quantity of urea in the vomited matters. Gréhant, who observed this last-mentioned fact, was unable to find more urea in the blood than under normal conditions. The history of hysterical ischuria is closely bound with that of vomiting. The ischuria may be secondary to the latter or primary, and it is not improbable that both troubles may be developed simultaneously. The ischuria may be temporary or permanent. Temporary and isolated ischuria often passes unperceived, and may hardly attract the attention even of the patient. It often follows a convulsive or other paroxysm. Permanent ischuria, although much less frequent, is more interesting. It may occur alone or be accompanied by compensatory phenomena, such as vomiting, profuse sweats, salivation, or diarrhœa.

Under varying circumstances a woman ceases to urinate. At the end of twenty-four hours she becomes disquieted; a catheter is passed,

but nothing is found in the bladder, and the latter also, as far as we can judge from palpation, is not distended. At the end of two or three days, if the anuria is complete, we see the occurrence of vomiting, either spontaneous or provoked by the ingestion of no matter how small a quantity of food. It has not been shown that the vomited matter ever has the odor of urine, or that the vomiting may be truly regarded as supplementary to the suppression of urinary secretion. The matters contain urea, as do, moreover, all vomited matters (Bouchard). In general, however, the amount of vomited matter increases in proportion as the secretion of urine diminishes. Sometimes the vomiting is replaced by diarrhœa or sialorrhœa. It would appear as though there were a supplementary elimination of the fluids, if not of the solid constituents of the urine. Ischuria associated with vomiting may continue for months; the latter, indeed, causes disturbances of nutrition and emaciation, but everything returns to the normal as soon as the excretion of urine is reëstablished. The cure is sometimes retarded by an habitual anorexia.

When the anuria is not compensated for by any other elimination, which is rarely the case, it can hardly last more than a few days. If it does continue longer, we may see the supervention of uræmic symptoms, such as cephalalgia, nausea, visual disturbances, and dyspnœa; but these manifestations are not necessary, for a patient of Holst was anuric for seventeen days without the appearance of any of these symptoms.

The Bladder.—The disorders of urinary excretion are much more frequently troublesome than those of secretion. Disturbances of excretion may be associated with sensory troubles of the bladder. Hysterical patients may suffer from vesical pains, either permanent or recurring in paroxysms, or permanent with paroxysmal recrudescences, and often in relation with uterine neuralgia and dysmenorrhœa. These neuralgias are often associated with dysæsthesia of the vesical mucous membrane, as determined by pressure on the abdomen or the introduction of a sound. This dysæsthesia is sometimes sufficiently intense to be a point of departure for an attack when the mucous membrane has been mechanically irritated, and it causes a continual desire to urinate. When the neck of the bladder takes part in the reaction we have retention of urine. Calculus may be simulated by hysterical cystalgia (Morand).

When the vesical mucous membrane is anæsthetic, contact of the urine no longer excites a desire to micturate, and distention occurs which may result in paralysis. This retention of urine is accompanied by incontinence from overfilling, unless care is taken to empty the bladder with a catheter. Incontinence, which is rare during an hysterical

attack, may be associated with spasmodic paraplegia, and may be due itself to a permanent spasm of the walls of the bladder.

Retention is very much more frequent than incontinence, with which it may alternate. It may be due either to contracture of the neck or to paralysis of the bladder. The introduction of a catheter will serve to differentiate the condition. Exploration by the catheter is the more useful, as the retention may coincide with ischuria. Retention is often temporary, but it may continue for some time, even for months or years, being complicated then by incontinence if we do not resort to catheterization. When retention is chronic the urine may become altered, and we may then see the ordinary consequences of this complication. Even death may follow (Brodie). Recovery may be sudden, however long the condition has lasted, and relapses are not uncommon.

The Urethra.—While the irritable bladder is often an isolated manifestation of hysteria, an analogous phenomenon may be observed in the urethra. In man the urethra is often the seat of spasms, either painful or painless, and I have long thought that these spasmodic strictures might be regarded as a local manifestation of hysteria. The passage of a sound to the deeper portions of the urethra may in these cases excite a convulsive attack (Guyon) or hypnotic phenomena (Boyd). Anæsthesia of the urethra is quite frequent, and when it is very marked it is not without influence upon the desire to pass urine.

Disorders of the Genital Organs.

The genital organs are often the seat of disorders of general and special sensibility. The testicles are frequently the seat of dysæsthesia, and pressure on them, as on the ovaries, may excite a convulsive attack. More rarely the testicles are anæsthetic, and pressure on them does not excite the ordinary testicular sensation. Sensory troubles of the testicle often coincide with anomalies of size, location, and direction; most frequently the testicle is small, sometimes it is arrested at the ring, and sometimes it is inverted with or without an anomaly of the epididymis. In women the uterus, as the ovaries, may be the seat of a neuralgic condition, with pain radiating in various directions. These pains are usually permanent, with paroxysmal recrudescences. The vulvovaginal mucous membrane may also be the seat of dysæsthesia or anæsthesia. The dysæsthesia is sometimes accompanied by local spasms, vaginismus, and often associated with spasm of the anal sphincter.

Hysteralgia is sometimes accompanied by menorrhagia or metrorrhagia, or these abnormal flows may occur without pain. The losses

of blood merit a special consideration because of the anæmia which they increase. We must admit, however, that amenorrhœa is more common.

The pollutions or secretions which sometimes accompany certain attacks with erotic characters may also be observed independently of them. These fluxes of glairy matters, of uncertain nature and origin, are seen frequently in connection with emotions, physical shocks, or fatigue, independently of any erotic manifestations.

The genital instinct is often very poorly developed in hysterical male subjects, and hysterical women are far from deserving the reputation for eroticism with which they have been credited. A great many of the latter, even when there is no marked local anæsthesia, never experience any sexual pleasure, despite the variety and the persistence of their attempts to secure it. This impotence, which is common to both sexes in hysteria, is the source of multiple sexual perversions, and sometimes of an acquired sexual inversion.

The Nature of Hysteria.

Notwithstanding the disdain which practice often affects for theory, it generally has to submit to its dominion. Practice, indeed, can reach its end only when it directs its efforts in conformity with the laws which are the objects of pursuit of theory, namely, the laws of the relations of facts. Whenever theory discovers probable relationships, practice follows; and even when the latter does not attain its end it seldom modifies its course, except under the influence of a new theory. In reality practice always originates from a theory, and it follows where the latter points. We see this in the history of most diseases, and also in that of neuroses and of hysteria in particular.

In default of explanations based upon positive notions, the neuroses have been from all time the object of theoretical speculations relative to their nature. Hysteria, which presents itself under such varied and variable aspects that we are accustomed to regard it as the Proteus of nosology, would lend itself particularly to hypothetical considerations. They have not been wanting, and they have had, according to the rule, a considerable influence upon the manner in which hysterical subjects and hysteria itself have been treated, both from a medical and from a social point of view.

According as they have been more carefully studied, the manifestations of this neurosis have multiplied and become diversified; and instead of regarding it as a morbid entity, many authors of standing tend to look upon it as a group of symptoms variously combined,

affecting the nutritive functions as well as those of relation, and with a variety that is infinite.

These combinations of symptoms, these syndromes as they are called, arise under an infinity of different circumstances. Without doubt they may arise by reason of an hereditary or congenital predisposition, as shown by a variety of stigmata. But the disease often appears, without any apparent original vice, as a sequel of acute or chronic diseases, infections, intoxications, or physical or moral shocks. Although these occasional causes act most frequently in awakening a hitherto dormant predisposition, and thus play the rôle only of exciting causes, yet in many cases we have to recognize that they determine themselves the troubles, by changing, as it were, the constitution. The morbid states which have been grouped under the name of traumatic neuroses, because they manifest themselves generally as a result of a railway accident, a stroke of lightning, or other violent shock, do not in themselves differ from the hysterical syndromes which may be observed in patients marked by the somatic stigmata of a morbid heredity or degeneration. It is the same with all the so-called symptomatic hysterias—that is to say, those which are developed in consequence of an anterior morbid state.

The variety of causes of the neurosis, the variety of its manifestations, and also their mobility and the frequency of their cure, would seem to excuse the pathologists for not having found a specific lesion, even if it were probable that one existed. The circumstances of the production of the affection show only that all congenital or acquired conditions of weakness favor it, whether these conditions are transitory or permanent. The possible action of blood changes is rendered probable by the fact that in chlorosis we see a number of troubles which might well belong to hysteria.

These uncertain data, most of which are, moreover, of very recent acquisition, could hardly serve as a base upon which to build a valid theory. For ages the opinions concerning the nature of hysteria have varied with the general ideas which might well be held concerning mental troubles. Hippocrates regarded insanity as a disease of the body, and it was impossible that he should think that it was the mind that was alone or primarily affected in hysteria. The peculiarly striking frequency of a group of determining conditions has served as a base for the theoretical localization of the primary lesion. The first observers were struck with the rôle which was played by the evolution of the genital function, by its episodes and its diseases, in the increase of the nervous troubles. A number of somatic and psychical manifestations, certain convulsive forms, and certain of the delirious hallucinations seemed to conspire to accuse the chief gene-

rative organ of being the seat of the disease, which was then regarded as peculiar to the female sex. The philosophers held the same opinion as the physicians. The uterus, they thought, is an animal which ardently desires to conceive; when it long remains sterile after puberty it can hardly bear it. It grows indignant and runs throughout the body, filling the tissues with air, stopping respiration, throwing the body into extreme danger, and occasioning various diseases.

The ancients regarded hysterical convulsions as something analogous to a consciously cynical and voluntary spasm. It was therefore a shameful manifestation. It was these ideas concerning the nature of hysteria which have given it such a bad, almost infamous, reputation. Freud ascribes to the early sexual experience a predominant rôle in the pathogeny of hysteria, but Pitres[67] does not regard it as important or frequent, and in this I agree with him. Among the most frequent methods of treatment was to advise marriage to girls and widows, and maternity to married women. This remedy, which was designed to satisfy desires which were frequently imaginary, served commonly to aggravate the evil.

Although certain forms of the convulsive attack may seem to have an equivocal signification, there is not the slightest doubt that most of the hysterical manifestations have nothing to do with the sexual instinct or its perversions. But the uterine theory had given currency to a prejudice which endured for ages, greatly to the detriment of the patients and contrary to good sense. The idea of infamy which, without any good reason, had been attached to hysteria, could only prevail at an epoch when renunciation of the sexual life had become a virtue. When, in the Middle Ages, under the influence of religious fears, hysteria manifested its contagiousness by many and prolonged epidemics, and declared itself under the theomaniacal and demonomaniacal forms, these manifestations often assumed the character of intellectual and moral anomalies or perversions, which led one to doubt its morbid nature. Hysterical subjects were regarded sometimes as saints, but more often as possessed of the devil, and were treated accordingly, without the possibility of any voice being raised in the name of science to protect them. Exorcisms and the stake have served more to the propagation of the disease than to its cure.

In reality the genital functions do play an important rôle in the production of hysteria, by reason of the place which they hold among the causes of the emotions.

After Jean Wier and his followers had succeeded in securing for hysterical persons the status of the sick, and after Lepois, Willis, and Sydenham had recognized that the neurosis was not confined to

women, the idea of the *furor uterinus* and its demoniacal origin had to be abandoned. Scepticism then took the place of the blind prejudices which had led too often to barbarous methods of treatment. During more than a century the neurosis was treated less severely, as was suitable for a morbid condition, to which was commonly given the name of vaporous disease. The "vapors" were the theme of an entire literature at the end of the last century and the beginning of this. What appeared the most clearly in the majority of these works was the predominating rôle of moral causes in the determination of hysterical troubles. This idea, that we had to do with vapors, paved the way for Mesmer's success; but, on the other hand, it could be sustained only by the success largely cried up of the new medicine. The old aphorism, "Cures demonstrate the nature of diseases," could not deceive. If, then, the vapors could be cured by medicine of the imagination, it showed that they were a disease of the imagination. And people did not well distinguish at this time, or even later, between diseases of the imagination and imaginary diseases. Furthermore, during the first third of this century, many authors, and some of them the best, regarded hysteria and hypochondriasis as one and the same affection; and this meant that the one as well as the other was a disease of the spirit. To-day an assimilation of the two diseases would have a very different signification.

Under the influence of the doctrines of Broussais a search was again made for organic lesions of the genital organs; several authors inclined to the ancient opinion; others attached more importance to the lesions of the ovaries, and it was chiefly from the works of Schutzenberger that the ovarian theory of hysteria took its origin; and to this Négrier later brought the weight of his observations. The almost constant presence of a pain in the region of the ovary, and usually actually located in the ovary itself, gave some probability to this theory. The recent study of the periodical functions of the ovary added still more to this probability because of the periodicity of certain hysterical troubles. Although the theory, combated by Briquet, soon ceased to enjoy any credit among physicians, it persisted longer in the minds of surgeons and even to within recent times has served as a basis for interventions which were regarded as radical; the ovary being taken away the hysteria ought to be cured. But experience has shown that the ovary being taken away and even both ovaries being taken away, the hysteria remains and sometimes finds itself enriched by mental troubles which did not exist before the operation.

Ch. Lepois and Willis believed, indeed, that they had discovered cerebral lesions in post-mortem examinations of hysterical subjects;

but their facts were not very precise and hardly deserve credit. It is to Briquet that the merit belongs of having recognized hysteria distinctly as an affection of the nervous system, a cerebral neurosis. Clinical studies gave a solid foundation to this opinion by showing that the sensory and motor troubles most commonly seen in hysteria had numerous points of resemblance to those due to cerebral lesions. The mobile character of these troubles in hysteria could be explained by a mechanism analogous to that which is employed to explain the motor and sensory troubles of certain forms of migraine, namely, by vascular spasms or by nutritive troubles of the cerebral cells dependent upon general conditions of the organism, themselves subject to multiple oscillations.

The regular study of the hypnotic phenomena undertaken at the Salpêtrière by Charcot and his pupils had helped to add greater weight to the cerebral theory. Certain unexpected associations in the paralyses from suggestion, such as that of the loss of articular speech accompanying a paralysis of the right upper extremity, and reproduction of the associations which are frequent in spontaneous lesions of the cortex of the left cerebral hemisphere, had brought into favor the hypothesis of the localization of hysterical troubles in the cortical layer of the cerebral hemispheres. The question was hardly raised as to whether there was a relation of coincidence or of succession between the idea of paralysis or of insensibility and the disturbance of nutrition of the cortex which appeared to be the indispensable anatomical condition in the production of peripheral paralyses.

Experimental hypnotism, in showing the rôle of the idea in the evolution of a great number of hysterical manifestations, had made manifest the rôle of the imagination in the pathogenesis of hysteria. The School of Nancy especially has taken an important part in this evolution of the theory of hysteria. According to these teachings all hysterical manifestations become the product of the imagination; they may be cured by everything that acts upon the imagination; the medicine of the imagination is not only the best, but is the only rational one when we have to do with a neurosis. We approach here the mediæval theory and its practical application; the evil spirit is no longer to be driven away by exorcism, but the pathogenic idea is to be driven away by suggestion. Hysteria has become a disease by representation (Möbius, Strümpell), a psychical malady *par excellence* (Charcot). Its different phenomena are very simply explained by a weakening of the psychological synthesis (Janet), by a narrowing of the field of consciousness, by a narrowing of the field of attention, of perception, of motor impulse (Pick).

These theories of a primary mental lesion might derive some sup-

port from a certain number of undoubted facts in which the sensory and motor paralyses, the dysæsthesias, the amnesias of hysteria do not correspond to the ordinary form of the same troubles when they are in relation with lesions of the cerebral cortex. We observe, in effect, in the neuroses facts in which the distribution of the troubles and the symptomatic association are manifested rather according to popular notions than according to known physiological laws. For example, there are described, as belonging exclusively to hysteria, the systematization of paralysis for a special movement, to the exclusion of other movements executed by the same muscles, and the superposition of sensory on motor troubles, and the superposition of disorders of sensation and of disturbances of function of an organ of special sense, such as are never found in the case of cerebral lesions.

However, a like superposition is sometimes observed as a consequence of lesions of the brain; in certain cases of a unilateral loss of sight the teguments of the eye are frequently affected just as in hysterical amblyopia. And on the other hand the systematization of certain hysterical paralyses is accepted without sufficient proof.

Another theory allied to the mental theories of hysteria is that which assumes that when an idea or the forced suppression of an idea has entered the mind it remains there as a foreign body, and keeps up a permanent irritation which may perpetuate accidents of the most varied sort from more or less dominating notions to convulsive attacks; and as long as the idea has not lost its emotional power it remains a cause of the awakening of the representations which play an important rôle as prelude, and perhaps as cause of the explosion of the motor or delirious paroxysms. This theory (of Breuer and Freud) supposes that the idea acts through the emotion, the rôle of which had already been well recognized by Briquet. The idea cannot act in this way except on the condition that the individual has a morbid emotivity; but emotion, whether normal or pathological, is indissolubly joined to physical conditions.

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If an idea is but a feeble sensation, a representation, we can hardly *a priori* admit that it is capable of acting in the pathogenesis of hysteria to the exclusion of physical agents, the action of which is at the root of representations. But observation shows that physical agents play a predominating rôle in this pathogenesis, in proof of which we may mention traumatic and toxic hysteria. The hypothesis that hysteria is a disease of the mind is opposed also by the same reasons which may be brought against the existence of disease of the mind in general.

The mind is not an organ, it is a function. Just as the function of digestion is troubled not only when its chief organ, the stomach, is affected by lesions of its structure, but also when the innervation, the circulation, and the quality of the blood which this organ receives are altered under the influence of general morbid conditions or even of diseases of other organs: so the mind, of which the brain is the organ *par excellence*, is affected not only when the brain is disordered by lesions proper to it, but also whenever the vitality of the brain is modified by troubles of nutrition, whether their cause be general or local. Properly speaking, there are no diseases of the mind, but the mind is troubled by all the diseases of the body which have as a consequence a disturbance of the functions of the brain. This is a conception which goes back to Hippocratic times but which was afterwards obscured by considerations which have nothing to do with science. From a practical as well as from a scientific point of view it is important not to forget this; for if physicians who treat insanity can congratulate themselves upon effecting a cure, it is not to what they know of psychology but rather to what they know of general pathology that they owe their success.

As we follow them through the animal scale, the manifestations of the mind appear to us like the products of irritation; their development marches in a parallel line with the development of the specialization of irritability, with the perfectioning of motility and sensibility and of their organs. No movement, no sensation, no mind.

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Apart from these movements, however, and even admitting that the rule of the isolation of the neurons is not an absolute one, the fact alone of the increase in number and in length of the fine ramifications of the nerve cell after birth may explain not only permanent acquisitions but also the more or less rapid oscillations of cerebral activity. Following the section of a peripheral nerve, the atrophic degeneration of the nerve fibre begins at the extremity farthest removed from the cell, which plays a trophic rôle. We may assume that the disturbances of nutrition obey the same laws in the brain, and that when a nutritive modification, however slight, occurs in the brain cell, it is its finest and latest developed prolongations which suffer the first. This supposition accords with the course of the process of dissolution in the mental organization; we know that in senile involution the latest acquisitions are the ones which are lost the first, and the same is true of the acute processes of dissolution; when, under the influence of a traumatic or moral shock, a partial loss of memory occurs, we find that it is the memory of recent events that has disappeared. Furthermore, it is in the most delicate portions of the tufts of the pyramidal cells of the cerebral cortex that the moniliform swelling (Andriezen), which seems to constitute the anatomical substratum of the toxic dissolution in alcoholism, is seen. The atrophic changes which have been met with in progressive general paralysis occupy the same seat at first, and they appear to follow, as regards the body of the cell, a centripetal course.[62]

On the other hand, in order that there may be produced an alteration in the nerve cells, it is not necessary that the organism be affected with very profound disturbances of nutrition, with an infection or an intoxication which may threaten life. The experiments of Sadowsky, of Hodge, and of others[63] show that, under the influence of fatigue, the nerve cells undergo important modifications, especially the nu-

cleus which becomes darker and deformed. As the conditions of fatigue may be realized in a great number of circumstances, we have reason to suppose that these circumstances are capable of provoking analogous cell changes and consequently modifications of conduction in the finest branches and in those farthest removed from the trophic centre.

Under the influence of fatigue the nerve activity is weakened in all its forms. Cutaneous sensibility is diminished, and the same is true of the special senses, the visual field is narrowed, the visual as well as the auditory acuity is blunted, perception becomes slowed, the motor reactions become weaker, slower, and less precise; the memory of faces and association become weak, the capacity of the consciousness diminishes, and the last manifestations of the nervous activity and those which are the most complex suffer earlier and more than the others. All the conditions which are capable of causing fatigue or exhaustion, physical or mental suffering, bad air, cold, or an insufficiency of food or of physiological stimulants may cause these same troubles. All these troubles which one could imagine in representing to himself the effect of a diminution of conductivity in the fibrils of the nerve cell are found in the physiological condition in the prodromic period of sleep, and pathologically in all the conditions of a lowered nervous activity, especially in the manifestation of hysteria, which resembles fatigue so closely that it might be compared to a chronic fatigue, from the point of view of its mode of production as well as from that of its biological characters. If we will recall the rapidity with which physical agents as well as moral influences act upon the nutritive functions, we may explain, through the mechanism of the neurons, the mobility of the manifestations of hysteria and of somnambulism, as also the rapidity of the passage from sleep to the waking state and from waking to sleep. All the exciting causes of hysteria and of the nervous troubles which resemble it appear to act in an analogous fashion; observation shows, indeed, that no form of morbid trouble of this category corresponds exclusively to a given cause, and that, apart from those which act purely locally, any one of the usual causes may produce the most diverse troubles. That which effects the specialization of the troubles is the nervous constitution of the individual which is often revealed by peculiarities of temperament or character. The morbid localizations may be attributed often, leaving out of consideration local shocks, to anomalies or retarded development either of the peripheral organs or of certain regions of the brain which remain throughout life sensitive points and easily fatigued. Numerous facts connecting teratology with pathology teach us that congenital defects are personal factors

of the greatest importance in disease. In hysteria especially the functional troubles are often localized on the side on which there exist chiefly or exclusively more or less evident morphological anomalies.

The theoretical mechanism, which may serve to explain the development of hysterical accidents, may also serve to explain the mode of their cure. It is the restoration of the nutrition of the cellular elements which is the basis of the restoration of function. This restoration is always dependent upon physical conditions. The cure produced by suggestion, by the idea, necessitates in general the intermediary of an emotional state, which is necessarily accompanied by organic change. The persistence of the cure is subordinate to the permanence of the modification of nutrition, which permits the nerve cells to resume definitely their normal function.

Just as we know that under the influence of a continued activity the nerve elements may take on a predominant development, which is shown by a permanent functional exaltation, so under the influence of a long-continued deficient activity the elements suffer the consequences of inaction. Furthermore, to the effects of a want of use there is added another element in the pathogenesis, namely, the combat between the different parts of the organism, which, as Weigert holds, consists in the fact that the healthy histological elements develop at the expense of the weakened or altered ones, and tend to take their place. This mechanism may enable us to understand how troubles which are indubitably produced by causes which have ceased to exist, and the effects of which upon nutrition appear to have been compensated for, may pass into the chronic state and persist indefinitely. The influence of some fugitive cause—a fatigue, a traumatism, or an emotion—may determine permanent results, which, despite appearances, have an anatomical substratum.

In fine, the new notions which we have acquired concerning the morphology of the cerebral cells give a weighty support to the corticocerebral theory of hysteria and of neuroses in general. They enable us to understand better the forms and the progress of these troubles, the influence which physical agents may exercise upon them, and, consequently, the purely secondary value of the representations, the ideas. We must recognize also that this old notion has never been completely obscured; for even in the best days of hypnotism and suggestion most medical authorities have opposed the applications of the medicine of the imagination. It was soon remarked that the experimental crimes suggested to hypnotic subjects were successful only when they were not in opposition to the tendencies of the subjects themselves. While from a medicolegal point of view we must recognize that we cannot apply to true crimes the interpretations in-

spired by the crimes of the laboratory, the same distinctions hold in a therapeutic sense; suggestion as a therapeutic measure has its restricted limits. Parallel with the anatomical theory of neuroses we have seen arise the practice of physical treatment, forced rest, and superalimentation brought into repute by Weir Mitchell. Practice directs its efforts in conformity with the laws imposed upon it by theory. But we have seen that this coincidence cannot constitute a proof of the truth of the theory at the time prevailing. Time only can furnish the proof of the truth or falsity of this theory.

Prophylaxis.

The frequency of heredity in the causation of hysteria and in its relations with degeneration would suffice to establish, in the absence of other facts, the frequency of a congenital predisposition in its development. To this predisposition we may oppose prophylactic measures, which ought to be instituted at an early age. Education in the family is in some sort a continuation of gestation; in morbid families this education produces an aggravation of the heredity. The child should be therefore removed as soon as possible from these morbid family influences; he should be given to a strange nurse, who has none of the neuropathic tendencies of the mother and none of her morbid emotivity.

The spirit of discipline should preside over the education of children predisposed to hysteria, and they should be educated in company with other children of their own age. We must guard them from the occasions of violent emotions and from causes of worry, but not so as to let them remain ignorant of the fact that the world is not made for their special benefit, and that they cannot hope for the complete realization of all their desires, and that they will obtain concessions only in proportion as they know how to give them. We must endeavor to develop in them altruistic sentiments, and to familiarize them early with the expression of these sentiments. A habit of becoming expressions, gestures, and attitudes necessarily determines becoming sentiments. Whatever may be later the development of egoism which is so common in these patients, this education will at least have the advantage of weakening the manifestations of this egoism. Self-control is much more speedily attained outside the family environment.

Burton closes his "Anatomy of Melancholy," with this recommendation: "Be not solitary, be not idle." This is a counsel which is applicable to those predisposed to hysteria at every period of their existence. The presence of strangers or of acquaintances presents an

port from a certain number of undoubted facts in which the sensory and motor paralyses, the dysæsthesias, the amnesias of hysteria do not correspond to the ordinary form of the same troubles when they are in relation with lesions of the cerebral cortex. We observe, in effect, in the neuroses facts in which the distribution of the troubles and the symptomatic association are manifested rather according to popular notions than according to known physiological laws. For example, there are described, as belonging exclusively to hysteria, the systematization of paralysis for a special movement, to the exclusion of other movements executed by the same muscles, and the superposition of sensory on motor troubles, and the superposition of disorders of sensation and of disturbances of function of an organ of special sense, such as are never found in the case of cerebral lesions.

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On the other hand, in order that there may be produced an alteration in the nerve cells, it is not necessary that the organism be affected with very profound disturbances of nutrition, with an infection or an intoxication which may threaten life. The experiments of Sadowsky, of Hodge, and of others[63] show that, under the influence of fatigue, the nerve cells undergo important modifications, especially the nu-

cleus which becomes darker and deformed. As the conditions of fatigue may be realized in a great number of circumstances, we have reason to suppose that these circumstances are capable of provoking analogous cell changes and consequently modifications of conduction in the finest branches and in those farthest removed from the trophic centre.

Under the influence of fatigue the nerve activity is weakened in all its forms. Cutaneous sensibility is diminished, and the same is true of the special senses, the visual field is narrowed, the visual as well as the auditory acuity is blunted, perception becomes slowed, the motor reactions become weaker, slower, and less precise; the memory of faces and association become weak, the capacity of the consciousness diminishes, and the last manifestations of the nervous activity and those which are the most complex suffer earlier and more than the others. All the conditions which are capable of causing fatigue or exhaustion, physical or mental suffering, bad air, cold, or an insufficiency of food or of physiological stimulants may cause these same troubles. All these troubles which one could imagine in representing to himself the effect of a diminution of conductivity in the fibrils of the nerve cell are found in the physiological condition in the prodromic period of sleep, and pathologically in all the conditions of a lowered nervous activity, especially in the manifestation of hysteria, which resembles fatigue so closely that it might be compared to a chronic fatigue, from the point of view of its mode of production as well as from that of its biological characters. If we will recall the rapidity with which physical agents as well as moral influences act upon the nutritive functions, we may explain, through the mechanism of the neurons, the mobility of the manifestations of hysteria and of somnambulism, as also the rapidity of the passage from sleep to the waking state and from waking to sleep. All the exciting causes of hysteria and of the nervous troubles which resemble it appear to act in an analogous fashion; observation shows, indeed, that no form of morbid trouble of this category corresponds exclusively to a given cause, and that, apart from those which act purely locally, any one of the usual causes may produce the most diverse troubles. That which effects the specialization of the troubles is the nervous constitution of the individual which is often revealed by peculiarities of temperament or character. The morbid localizations may be attributed often, leaving out of consideration local shocks, to anomalies or retarded development either of the peripheral organs or of certain regions of the brain which remain throughout life sensitive points and easily fatigued. Numerous facts connecting teratology with pathology teach us that congenital defects are personal factors

useful hygienic measures. When exercise is impossible by reason of the patient's morbid condition, passive movements, Swedish gymnastics, and massage may be of great assistance. During bad weather, or when the sensitiveness of the patient or some complication renders it impossible for him to go out in the air and sunshine, inhalations of oxygen may be of assistance. These often have a very happy effect upon the appetite, and they are very useful for the relief of special symptoms, such as anorexia and vomiting.

Hydrotherapy justly occupies an important place in the general treatment of hysteria; the vascular reactions which it produces constitute a veritable gymnastics of the skin, the activity of which influences all the functions. Cold water has much the most intense action, and it is that which renders the greatest service. Hot water in the form of douches or baths is indicated only under exceptional circumstances, when we have to combat a too intense excitation or a violent delirium.

The local application of cold water, and especially of ice, may be utilized in the treatment of localized pains and of hysterical zones, of which it may cause the transference or even the complete disappearance.

Cold water is more often employed in the form of general douches. It causes a general contraction of the cutaneous vessels, a contraction soon followed by a dilatation, which constitutes the reaction. Corresponding to the changes in calibre of the vessels and to the modification of the circulation resulting therefrom, we note a modification of sensibility. This is increased during the period of reaction, and absorption is favored (Fleury), together with the other phenomena of nutrition (Quinquaud). The utility of cold water is directly dependent upon the reaction which follows its application.

The douche acts by the force of percussion as well as by the temperature of the water. These two actions may in a certain measure compensate each other; the mechanical effects of the shock may compensate in part the elevation of temperature. The most efficacious douche is the cold douche with broken jet and of short duration. The pressure should be equivalent to that of about one atmosphere or one atmosphere and a half; its force may be moderated by breaking the jet with the hand or with a suitable spatula. The duration of the douche varies inversely as its force and the coldness of the water, and it should be quite short if the water is very cold and under strong pressure. The temperature of the water should, as far as possible, be uniform. If it is 7° or 8° C. (44°–46° F.), for example, the douche will last fifteen seconds at the most; but if the temperature gradually rises to 10° or 12° C. (49.5°–53° F.), we may prolong the douche to

twenty or thirty seconds. The douche should be general, avoiding the head and letting the water run down the lower extremities and feet, so as to facilitate reaction. We should avoid too strong and localized percussion, especially when there are hysterogenic zones which might be excited by the shock. We may be able to modify the sensibility of these zones by approaching them gently and gradually.

The cold douche is not equally well tolerated by all patients. There are some in whom we can obtain no reaction, even on increasing the pressure and using the coldest possible water. We may often remedy this inconvenience by following the douche with dry rubbing by means of the bath glove. But if reaction does not follow even then, we must renounce the use of cold water. More often the cold water causes fatigue and an exaggeration of the nervous troubles; but as long as it is followed by a reaction and does not provoke new troubles proper to it, we must persevere, and often at the end of some weeks the good effects will be manifested. Cold douches may in themselves determine certain troubles, especially headache and vertigo. But this may be avoided by keeping the head covered with a cloth wet in cold water while the douche is being given, and in following it by a vigorous slapping of the soles of the feet and lower part of the legs, and following this by a hot footbath. At other times the cold douche causes dyspnœa and palpitations, which generally become less marked as the patient becomes accustomed to them. We may combat them, however, by the application to the chest of a cloth wet in cold water, and in avoiding the direction of the douche upon the anterior thoracic region.

When the cold douche is not well borne, either because the reaction is absent or because it is followed by phenomena of excitation, we may resort to the Scotch douche, which is made by means of the watering pot employed in two different ways. The Scotch douche without transition is one in which warm water at a temperature of 35° to 45° C., according to the tolerance of the patient, is sprinkled on him for thirty to one hundred and eighty seconds; then cold water immediately follows for a period of five to fifteen seconds. This procedure, which may be useful in other morbid conditions coinciding rather commonly with rheumatism, sometimes causes excitation. The extreme temperatures may not be better supported than the rapid transition. In this case we may resort to the Scotch douche with transition, in which the temperature of the warm water is gradually lowered and in which we often reach a cold temperature only after several douches. This transition method enables us to pass gradually to the use of the pure cold douche.

Hydrotherapy without percussion plays a much less important

rôle in the treatment of hysteria. The cold bath (7° to 8° C.—44° to 46° F.) in running water is generally badly borne even for two or three minutes; sometimes it causes very energetic reactions which are not to be despised. The cold still bath (12° to 15° C.—53°-58.5° F.) is often well borne for a period of ten to twenty seconds, under the form of immersion, during which the patient should not remain motionless. As a rule, the stay in the bath should not be prolonged, although when the effects of the cold are not felt we should encourage an hysterical patient with paralysis to attempt a few movements which are made better in water, because of the loss of weight; and if he is successful this may have an important psychical influence.

Like the douche, the tub bath should be followed by a reaction, which may be favored by a walk or exercise taken before the bath, but which should not be so energetic as to cause perspiration, shortness of breath, or increased heart action. The emersion may be followed by a walk or some form of moderate exercise. In patients who cannot walk, massage, passive movements, or hot applications may take the place of the exercise during the first few days; but if the spontaneous reaction continues to be wanting, we must give up the use of cold water.

Whatever the form of hydrotherapy adopted, these measures should be followed out with constancy, regularity, and a military exactitude. Moreover, punctuality in the taking of food, in exercise, in sleep, and in other hygienic measures, should be an absolute rule in neuropathic patients, and especially in the hysterical. The douche should therefore be taken every day, exactly at the same hour; and the repetition of this measure every day is of advantage not only from the point of view of vascular exercise, but also from that of general discipline. If but one douche a day can be taken, it should be taken in the morning as soon as possible after waking; it may follow the first breakfast without inconvenience, especially if the distance of the patient's residence from the hydropathic establishment necessitates a walk of ten or fifteen minutes. The second douche should be taken as soon as possible before the evening meal. In general, the cold douches have no injurious effect during the menstrual period; it is more prudent, however, to continue the hydrotherapeutic measures only during the second menstrual period after the treatment has been begun, and to intermit them during the first.

The regular practice of hydrotherapy necessitates not only special apparatus, but also a suitable locality. The cold douche should be taken in a warm and dry apartment, and it is not a matter of indifference whether this room be well lighted or not. Patients who are easily depressed do not willingly enter naked into a damp, dark,

and cold cellar, and we should have regard to their feelings in this respect. The douche is better borne when it is applied boldly without any hesitation. From these considerations we may deduce the fact that the arrangements in private houses are for the most part insufficient for the carrying out of these necessary measures. Whenever possible, the water treatment should be carried out in a medical establishment, in which the director, guided by special experience, carries out with authority and regularity the treatment decided upon in consultation with the family physician. But establishments of this kind which merit confidence are rare; and the physician should hold himself in readiness to direct personally the treatment in non-medical establishments, in which no other claim in made than that of willingness to carry out the physician's orders.

In the absence of the facilities afforded by special establishments or special apparatus, we are often obliged to have recourse to procedures which are less efficacious than the douche, but yet are of a certain value. Among these we may mention, first, the wet sheet, a thick cloth dipped in cold water and then wrung out. The patient, standing and naked, a damp sponge having just been passed over his face and chest, is wrapped in this sheet, which is pressed closely round his lower extremities, leaving the soles of the feet in contact with the floor. While the nurse rubs his back and limbs, he himself makes friction on the chest with the part of the sheet which covers it. This friction should be continued until the skin and the sheet itself are warm. The wet sheet is then replaced by a dry one, and the rubbing is continued with this. The reaction will be favored by a walk, or by massage if the patient can take no exercise. The wet sheet may be of service as a preliminary to the douche. If it causes oppression, we may limit its application to the lower portion of the body; if it causes headache or vertigo, we may remedy that, as in the case of the cold douche, by having the patient keep his feet in warm water during the operation.

The dripping sheet is applied like the preceding, but without having been previously wrung out. While the body is enveloped in the sheet, it is rapidly tapped all over until a certain degree of warmth is obtained, and then the wet sheet is replaced by a dry one and friction is made. These wet packs differ in their effect according to their duration. If they are made for twenty or thirty minutes, and if, at the time of reaction, a cool lotion is made and followed by a dry rubbing, they have a sedative and tonic action; and if made in the evening before retiring, they are very useful as a remedy for insomnia. If they are continued for one or two hours, they have a sudorific effect; followed by a cold affusion or douche, they have a tonic and resolvent effect.

Cold affusions and lotions, followed by frictions, are methods which may be employed in mild cases, but they are generally ineffectual in the graver forms of hysteria.

Although, as we have before said, we ought to advise the carrying out of hydrotherapy in medical establishments and under the supervision of a physician, there are nevertheless certain establishments with patented forms of apparatus against which we ought to be on our guard. There are institutions of this sort which are more concerned in keeping the patients for a long period within their walls than in curing them and filling their places with newcomers; and certain physicians, in order to display their aptitude for observing and acting upon the most subtle indications, modify almost every day the mode of application of an agent which depends for its success upon discipline and uniformity of application.

Among the physical agents utilized in the treatment of hysteria, electricity in its various forms holds an important place. Static electricity especially has won for itself, since the lessons of Charcot at the Salpêtrière, a right to be regarded as a valuable therapeutic measure. The electrical bath has physiological effects of varying intensity, according to the individual. It has a general tonic action; the capillary circulation is modified, the volume of the limbs increases, the cutaneous perspiration is more active, and the appetite is increased; sensation reappears in the anæsthetic regions, the dynamometer shows a notable diminution of the amyosthenia, the contractures often disappear, and the special activity of the hysterogenic zones greatly diminishes or entirely disappears. The effects are more or less durable, being lengthened in proportion as the séances are repeated, and finally becoming resolved into a permanent benefit to nutrition. In women the menstrual functions become regular, digestion grows less difficult, and the weight increases; finally, the patient's mental state undergoes corresponding improvement. The bath of static electricity should be repeated two or three times a week at regular intervals. We should begin with moderate tension and gradually increase it later. The tension should be diminished if the séances are seen to be followed by excitation. When the static bath has a marked sedative effect, it may, if administered in the evening, be very useful as a remedy for insomnia. If it is desired to act upon the local symptoms, such as the anæsthesias, the dysæsthesias, the hysterogenic zones, etc., we may, by means of suitable apparatus, produce an electrical breeze or a spark in the affected region.

Duchenne and Briquet employed faradic electricity exclusively in the treatment of hysteria. The faradic brush acts energetically upon the cutaneous sensibility, and it is very useful in local or generalized

anæsthesia. It acts also on the motility, but rather in the manner of the æsthesiogenic agents than by its special action on the muscles. Electrical baths and douches are but little used, and general faradization, given after the method of Beard and Rockwell, sometimes appears to replace with success the static bath. General faradization, practised two or three times a week, ought not to be given for more than five or six minutes at each séance. Galvanization is hardly justifiable, except in the case of trophic troubles, and especially of the amyotrophies, which are moreover quite rare.

Hydrotherapy and electricity accomplish so much in the treatment of hysteria that the metallotherapy of Burq and the employment of vibratory machines or tuning forks scarcely deserve more than a mention, although certain special conditions may present themselves in which these measures are not to be despised. Massage, passive movements, mechanotherapeutic apparatus, kinesitherapy, may be employed in certain special cases, upon a consideration of which it may be useful to dwell briefly.

Massage and passive movements have an important place in the Weir Mitchell plan of treatment, which, with its other essential elements, isolation, systematic repose, and overfeeding, is often indicated in the management of hysteria, especially in those cases in which the troubles have appeared in consequence of severe traumatism or of acute disease. We need not dwell here upon the details of this well-known method of treatment.

Apart from the systematic treatment of Weir Mitchell, overfeeding is often of service in hysteria, in which the diet should be carefully regulated. While combating as strongly as we can the caprices of appetite of these patients, it is better often to make certain sacrifices in order to satisfy their taste than to expose them to the risks of an insufficient dietary.

Sleep is the more necessary to the individual as he is the more feeble; in the healthy adult it favors the preservation of the strength, in the child it favors growth, and in the sick it promotes repair. In hysterical subjects the sleep, which is indispensable to a cure, is often disturbed, and therefore it is necessary to favor it by all the means which hygiene puts at our command. Exercise in the open air and regularity in the hours of retiring and rising must first of all be recommended. We must assure to the patients the most perfect tranquillity. We must also take care that the light is not so bright as to keep the patient awake, and to remember also that all impressions upon the senses are capable of preventing sleep. We should therefore avoid noise, strong odors, or irritant contact of any kind. Certain forms of food or of drugs which leave powerful gustatory

impressions may be a cause of insomnia. The ingestion of a small quantity of food, of a warm or slightly stimulant liquid, before retiring favors sleep. In cases in which the patients awake three or four hours after having gone to sleep, we may cause sleep to come again by giving them a little nourishment, such as a cup of bouillon or a glass of milk. These patients are very sensitive to cold, and this may prevent sleep. Many anæmic patients, and this includes almost all hysterical subjects, are somnolent during the day, because the anæmia of the brain is more pronounced in the upright position; but at night, when in an horizontal position, the organ receives an amount of blood to which it is not accustomed, and insomnia results. This insomnia yields only to general treatment, and for these patients iron is the best narcotic.

Even those who hold that hysteria is a psychical disease do not deny that iron and tonics are useful adjuncts to the treatment of the affection. We, on the other hand, do not deny, however, that bread pills and other equally inactive preparations may aid in the cure of hysteria; but we believe that these so-called psychical agents act by exciting emotions of which the necessary physical accompaniments are the equivalents of the physical effects of more substantial intérnal or external remedies. These latter agents deserve so well their preëminence that the most obstinate partisans of the psychical origin of hysteria do not hesitate to include in their therapeutic armamentarium certain grossly physical agents, such as cold water, for example. The influence upon hysteria of agents capable of modifying nutrition shows well that hysteria is under the dependence of somatic conditions, of which we must first of all take account. That is to say, that the medicinal treatment of hysteria is more important than many believe, since it is legitimately employed in a number of morbid conditions which play an important rôle in the etiology of hysterical troubles. Among such are intoxication from external sources, autointoxication, infection, anæmia, etc. In hysteria of the menopause, the treatment of arteriosclerosis or of obesity may cause the disappearance of nervous troubles, just as may the treatment of anæmia in the hysteria of puberty.

Treatment of the Individual Symptoms.

We have incidentally mentioned a certain number of measures which may be employed against the individual manifestations of hysteria, and there remains little for us to say on this subject.

The convulsive attacks which are the most striking manifestations of the disease, and at first sight the most alarming, are not really in themselves of any special gravity. We should limit ourselves to see-

ing that the patient does not wound himself, and to the reassurance of the family. Frequently the loss of consciousness is not absolute, and in this case numerous onlookers may only excite the patient. We should therefore permit the presence only of those persons who are indispensable, and among those select the ones whom the patient may regard with indifference; we should impose silence on these, should moderate the intensity of the light, and should restrain the patient in the simplest possible way, by means of a folded sheet placed transversely across his body. We are rarely forced to hold the limbs, and when this is necessary it should be done with care, by surrounding the wrists and legs with wadding before restraint is applied; and especially we should avoid any struggle in which the patient might be wounded or might wound others, or in any case be uselessly excited. When the attack is over we should not relax surveillance until after a certain period of calm has elapsed, for the attacks often occur in series; but we should avoid the maintenance of any restraining apparatus which would attract the attention of the patient. Compression on hysterogenic zones may cut short the attack, and various forms of apparatus have been devised for making pressure on certain points, over the ovary, for example; but in general, compression has only a postponing effect. Nevertheless, compression may serve to modify the form of the manifestation; and when we have to do with specially grave dyspnœic or delirious troubles, it may be of advantage to change the character of the attack. Hypnotism or inhalations of ether may cause a convulsive attack to cease, but they are often provocative of a delirious attack. Chloroform in fractional doses may be useful in putting an end to a spasm which causes anxiety or inconvenience. Hypodermic injections of morphine, which have been recommended in these cases, seldom produce the desired effect, and they may readily lead to the formation of the morphine habit.

Prolonged hypnotic slumber, which is in reality a form of hysterical paroxysm, may interrupt or supplant a convulsive or delirious attack. The patient should then be allowed to sleep sufficiently long to obtain the equivalent of a night's rest. It may be desirable to change a delirious, lethargic, or cataleptic condition into a convulsion, which offers greater chances of a speedy termination. The stimulation of hysterogenic zones may enable us to obtain this result.

The dysæsthetic zones often in themselves constitute symptoms sufficiently troublesome to demand special intervention. Briquet noticed that compression might relieve the cephalic clavus; and I have often made use of a compressive apparatus which gives temporary results. A cap weighted with shot has sometimes done me good

service. The dysæsthetic zones may in general be influenced by the faradic brush, which was employed by Briquet. Metallotherapy, the use of the magnet or of a vibratory apparatus, especially the tuning fork, may also produce a temporary cure, often after having produced a transference with consecutive oscillations. Static electricity and local refrigeration by means of ice or a spray or chloride of methyl, may produce the same effects.

Cutaneous irritation by means of the faradic brush, a metal brush, sinapisms, etc., has often a happy effect upon localized or generalized anæsthesia. A modification induced in the cutaneous anæsthetic plaques which cover organs affected with paralytic or spasmodic motor troubles, often produces a beneficial effect upon the latter. In this way we may obtain relief of aphonia, mutism, cough, pharyngeal spasms, etc. Hysterical deafness or amaurosis is often cured by faradization of the skin of the auricular or orbital region. Static electricity has often a happy effect upon hemiplegic or generalized anæsthesia, as well as upon the partial or general amyosthenia which accompanies it.

Contractures have a special tendency to continue for a long period of time, and we must therefore attack them at their beginning and prevent them from dragging along (Charcot). We should begin as soon as possible with frictions and with massage of the antagonistic muscles, if they are accessible. This is done easily when the contracture follows an attack suddenly, but if it is produced insidiously in an inaccessible region, as in the thoracic muscles or those of the hip, the contracture will have often existed a long time before it is discovered, and its treatment will then be proportionally difficult.

If there are painful points, faradization of the skin, the use of the metallic brush, or superficial massage may, by diminishing the pain, favor the resolution of the contracture. Often we are reduced to the production of hypnotic sleep or to chloroform narcosis. When these procedures effect only a temporary resolution, we may be forced to bring on a convulsive attack, which sometimes leaves the member free upon its subsidence. Chloroform, even when it does not effect a cure, enables us to establish the diagnosis, and to form an opinion relative to the benignity of the affection, an opinion which we may also impress upon the patient. Furthermore, this temporary resolution gives us time to apply a suitable apparatus, if the member happen to be in a vicious position. As we have seen, these vicious positions, in spite of the ulterior cure of the contracture, may become permanent by reason of the fibrotendinous retractions. These retractions may later necessitate surgical intervention, from which we may look for a perfect result (Charcot and Terrillon).

Although the treatment of the paralyses is less urgent than that of the contractures, we should not lose sight of the fact that the longer they have lasted the less amenable they are to treatment; and we ought, therefore, to intervene as early as possible. Static electricity, especially with local sparks, faradization by means of the brush, massage, frictions with a metallic brush, applications of æsthesiogenic agents, magnets, metals, etc., are often useful. Systematic exercises may play a particularly important rôle. These exercises have for their base two physiological facts: (1) The natural tendency to symmetry of the movements; and (2) the necessary accompaniment of the mental representation of a movement by a movement in miniature. The movements which are made with the free hand exercise the paralyzed hand in a certain measure. The sight of a movement which is executed for the patient, passive motions made upon him, and the repetition of even very feeble motions, are so many procedures for recalling the motor function. Not only sensory stimuli and the recall of motor images, but also mental stimulation, intellectual labor, are capable of aiding in the restoration. All these methods are accompanied by changes in the circulation, and consequently by functional changes. Trophic or vascular troubles, which accompany the paralyses and contractures, are amenable to general treatment. Like the contractures, the paralyses may lead to vicious positions of the members, which may call later for special treatment. We ought therefore to have a watch upon these commencing deformities, especially of the feet, where the tendency to equinus is increased by the pressure of the bedclothes.

The treatment of the visceral manifestations of hysteria is limited to meeting the general symptomatic indications. The troubles of phonation, dyspnœa, hæmoptysis, hæmatemesis, salivation, etc., scarcely furnish any special indications, unless they are accompanied by local sensory troubles upon which we may act by ordinary methods—æsthesiogenic agents, faradization, etc. Tympanites and borborygmi are sometimes favorably influenced by franklinization.

Anorexia calls first of all for isolation, the importance of which has been demonstrated by Charcot. Hydrotherapy constitutes a useful adjuvant, but well-regulated discipline is of first importance. The taking of food should be insisted upon the very first day, and if our commands are not efficacious we must resort without hesitation to feeding by a tube passed, whenever possible, through the nose. It sometimes happens that food which is vomited when voluntarily taken is retained when introduced through the tube. Nevertheless gavage ought to be employed only as a last resort, and whenever possible we must make use of the natural channels. We should search

with care for the existence of painful points, which may be in intimate relation with the gastric troubles; and, if found, combat them with appropriate remedies. We must also keep a watch on the patients to see that they do not yield to their tendency to force themselves to take violent exercise. In some cases inhalations of oxygen or baths of compressed air immediately before meals exercise a beneficial influence upon the appetite.

In my opinion suggestion in the treatment of hysteria is merely an accessory measure, the effects of which are most commonly temporary only; but in the case of anorexia it may sometimes be of great importance, since its effects, even though temporary, render alimentation possible, and thus exert a favorable influence upon the nutrition of the patient. The moral influences which we may bring to bear in the treatment of hysteria do not consist solely in suggestion, which has for its principal object the modification of ideas, but also in the provocation of tonic emotional states. Thus pleasant surroundings, the avoidance of annoyance, etc., are not indifferent measures in the treatment of anorexia any more than they are in that of the other manifestations of hysteria. This favorable influence of the emotional condition shows itself in a very evident manner in individuals who, after having suffered from a shock, had presented various symptoms of hysterotraumatism, and who finally obtain in court the damages which they had been striving after for months or years. These troubles, which continued and even increased as long as the process lasted, often improve or disappear, sometimes even with an astonishing rapidity, as soon as the damages are obtained. There is no question of simulation here; there is an emotional action, mental in appearance, but very physical in reality. These circumstances show in what measure it is important in the case of hysterotraumatism to seek to shorten and reduce to a minimum the patient's legal preoccupations. We may work in his interest by inducing him to compromise the case, and in that way put an end to the legal process and consequently shorten his malady.

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EPILEPSY.

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EPILEPSY.

General Remarks.

EPILEPSY has been known since the most remote antiquity. It has been called *morbus herculeus* because Hercules was affected with it, *morbus divinus* and *morbus sacer* probably because the Pythonesses writhed in epileptiform convulsions while they delivered their oracles. The first observers, struck by the suddenness with which the disease overwhelmed persons in perfect health, gave it the name which it still retains (from ἐπιλαμβάνειν, to seize). Among the Romans the name *morbus comitialis* was derived from the prevailing practice of suspending the assemblies (*comitia*) when a man was attacked by epilepsy during their progress. The disease has also been called *haut mal*, falling sickness, *mal de St. Jean*, etc.

Until recent times the disease has been included among the neuroses, that is to say, among the diseases of the nervous system with unknown cause. This fact is explained by the inconstancy of lesions observable at the autopsy upon such patients, and even in a large number of cases by the entire absence of gross or macroscopically visible lesions.

However, the observation of certain associated physiological or pathological states such as the puerperium and renal affections has started a division in the field of epilepsy. Accordingly, the eclampsia of pregnancy, that of renal disease, and infantile convulsions have been described separately. The convulsions which have been called epileptiform are, moreover, characterized by their appearance in the form of crises following each other rapidly in series. It is important to make this distinction, but it has been inspired by causal considerations or at least coincidences, rather than by symptomatic differences which in a large number of cases it is impossible to make out. They are indeed epilepsies; but they are acute epilepsies, supervening under the influence of occasional causes, such as pregnancy, childbirth, scarlatinal nephritis, etc.

DEFINITION.

As a matter of fact, we must look upon epilepsy as a group of symptoms' which may appear in greater or less number in the course

of very different pathological conditions, sometimes under one form, sometimes under another. To say that there is only one true, essential epilepsy which occurs without appreciable cause seems to me no more admissible than to claim that there is only one true angina pectoris which has for its cause contraction of the coronary arteries, and false anginas of toxic, hysterical, and other origin. These terms, epilepsy and angina, apply solely to a symptom complex; the most varied causes may produce the same clinical phenomena.

With this understanding I shall describe the epileptic symptoms in their different forms—motor, sensory and sensorial, visceral, and psychical. I shall indicate at the start how these symptoms, which are rarely isolated in a patient, may group themselves together.

Epilepsy may be general or partial, that is to say, it may manifest itself at once in the entire body or it may be restricted to a single part—to one half of the body, to one limb, or even to an isolated group of muscles. I shall begin with partial epilepsy.

Partial Epilepsy.

The term partial epilepsy is used to designate that symptomatic form in which the convulsions are limited to one half of the body or to a single limb, to the face, or to a group of muscles. It was described by Prichard in 1822 under the name of local convulsion or partial epilepsy, by Bravais in 1827 under the name of hemiplegic epilepsy, and by Elliotson in 1831 again under the name of partial epilepsy; but it has been more particularly studied in recent years by Hughlings Jackson, who has clearly recognized the interest, from a diagnostic point of view, which attaches to the location of the cerebral lesions.

Etiology.

It forms a portion of the symptomatic picture of acute or chronic forms of meningitis. Syphilis is one of the most important etiological factors, by the formation of neoplasms to which it often gives rise in the meninges or in the brain.

Epilepsy is observed after fractures of the cranium with depression and irritation of the cerebral cortex and therefore constitutes an urgent and precise indication for surgical interference. Sometimes it manifests itself a long time after the traumatism.

It may occur in persons previously or subsequently attacked by a hemiplegic or partial paralysis the result of a cerebral lesion of vascular origin. It may even be due to simple vasomotor disturbances,

as in certain forms of migraine. Uræmia figures among the causes of partial epilepsy, whether it acts directly by producing local œdemas or as an exciting cause in patients with disseminated lesions of the cord and brain. Finally, partial epilepsies of reflex origin have been described, whose point of origin is a peripheral irritation acting upon the nerves of the extremities, of the trunk, or of the viscera, particularly the pleura.

Special mention should be made of infantile hemiplegic epilepsy. It may be congenital or may manifest itself at a variable time after birth in persons who during infancy have suffered from convulsions followed by hemiplegia. No matter what may have been the origin of the latter, the onset of partial epilepsy is often the result of very ordinary causes, such as emotions, exertions, alcoholic excesses, and the like.

Symptoms.

The attack of partial epilepsy occurs often without any preliminary sensory or motor phenomena; but sometimes it is preceded by various sensations, such as pain, numbness, cold, etc., similar in every respect to the sensations described as the aura of epilepsy. The sensory auras generally affect the part which is liable to be the seat of the initial spasm. In one of Jean Charcot's cases the attack was preceded by a dream of being bitten. An interesting point in the history of these auras is that they may be transferred from one side to the other by means of a blister or ignipuncture (Hirt, Buzzard). The initial cry is absent, as a rule, and if the attack remains strictly partial there may be even no loss of consciousness—the patient is a spectator, as it were, of his own seizure. The spasm begins in the face or in an extremity, and extends hence according to certain rules determined by Hughlings Jackson.

When it starts in the face it may centre in the eye or in the mouth. The eye moves laterally or obliquely upwards; the eyelids twitch. The labial commissure is tremulously drawn upwards and outwards; the tongue is pushed forwards and twisted laterally. Then the spasm radiates to the rest of the face; the levator alæ nasi sometimes undergoes rhythmical contractions which impart to the features a peculiar grinning aspect. At times gnashing of the teeth is observed. Then the muscles of the neck are involved; the head is turned towards the side to which the eye and mouth deviate. If further extension of the spasm occurs the superior extremity is involved first, then the inferior extremity in its turn. When the attack affects the right side it is often preceded by aphasia (Jackson).

The spasm may begin in the upper extremity: the thumb and index finger are first flexed convulsively into the palm of the hand, and the other fingers follow with the same movement; the forearm muscles are violently agitated, then those of the arm and shoulder are involved; and finally the whole limb is tremblingly elevated. In this case the invasion extends first through the muscles of the neck and face, and later to those of the inferior extremity.

When the convulsion starts from the lower extremity it travels from below upwards, by commencing in the great toe, which is extended or flexed convulsively, and successively affects the leg and thigh muscles. While in the upper extremity movements of extension predominate, in the lower extremity flexion is chiefly noted. As in attacks of ordinary epilepsy, we may distinguish a tonic phase preceding the clonic phase; when loss of consciousness occurs the attack leaves behind a period of stertor which is generally short.

But the clinical picture varies greatly according to the more or less extensive spread of the spasm. When the convulsions remain limited to an arm, not only may consciousness continue intact, but the patient is often able to speak or to attend to some occupation during his attack. On the other hand the convulsions, beginning unilaterally, may invade the opposite side; the attack, at first partial, becomes general. In the latter event loss of consciousness is the rule, but it appears late, contrary to what occurs in general epilepsy of sudden onset—the patient witnesses the attack in its inception.

The period of stertor which follows the attack corresponds to the intensity of the latter and the degree of its spread. Subsequent muscular weakness is the rule and predominates in the affected muscles. Transitory or permanent paralyses are observed in the limbs in which the convulsions were intense and oft repeated, also disturbances of speech, stammering, paraphasia, and true aphasia. These paralyses are more frequent than after the greater crises of general epilepsy and are important from a diagnostic point of view.

As a rule the attacks of partial epilepsy in the same person always manifest the same form, start always from the same group of muscles, the same extremity, or at least the same side of the body. Complete attacks of partial epilepsy in general occur only at rather long intervals, of several weeks' or even several months' duration. At such intervening times minor incomplete attacks, aborted crises, often take place. Sometimes, however, the paroxysms recur more frequently, even daily. They may also manifest themselves in serial form, as the status epilepticus, and terminate fatally.

Sometimes the attacks are replaced by a series of shocks, or by limited spasms often associated with general excitement. At other

times their place is taken by fits of trembling which may last several days. Occasionally these shocks continue and extend over the entire half of the body; for such cases Koshevnikov has proposed the name of continuous partial epilepsy.

In connection with partial hemiplegic epilepsy which I have just described Charcot distinguishes two special forms which he terms tonic partial epilepsy or partial epilepsy with contracture, and partial vibratory epilepsy.

In *partial tonic epilepsy* the muscles of the neck contract on one side, drawing the head towards the corresponding shoulder. The arm contracts in a position of extension and pronation; the hand is twisted and placed at a right angle to the forearm and is applied across the dorsolumbar region, while the patient bends his body towards the contracted side. At the end of some seconds or minutes the patient bends in the opposite direction and the attack is over. This form rarely presents itself independently of every other form, but it may be frequently observed alternating with attacks of ordinary partial epilepsy.

Partial vibratory epilepsy is characterized by tetanoid shocks which affect the limbs contractured in extension, during the tonic phase; then follows a period of clonic convulsions in the same regions. Loss of consciousness occurs sometimes, especially when the facial convulsions are very intense. Occasionally a short period of stertor ensues.

Infantile hemiplegic epilepsy presents only one other special character in that it develops in persons affected with hemiplegia which dates from infancy and is known by the name of infantile spastic hemiplegia. The latter appears at that early age often as a sequel of violent and repeated convulsions and after a variable time it is associated with contracture. Partial epilepsy is not a necessary concomitant, but it is very frequent in patients thus affected. The attacks usually appear rather early, though sometimes not until several years later.

The convulsive attack in these patients does not differ from the one just described. As a rule the onset is preceded by an aura or some sort of premonitory sensation. The initial cry is not always lacking, but it is not generally the spasmodic cry of ordinary epilepsy; often it is a cry of surprise or pain of which the patient is conscious. Usually the convulsions remain limited to the paralyzed side or at least they always predominate on that side when they become general. Involuntary micturition is exceptional; the tongue is rarely bitten. The period of stertor if it occurs is usually short; the subsequent delirium remains absent as a rule. The attacks may ensue in series,

one paroxysm may follow before the preceding has terminated, and thus constitute a veritable status epilepticus. Death may result under such circumstances, even in the midst of convulsions which are strictly hemiplegic.

Partial epilepsy of infancy may be associated with other concomitant disturbances, such as shock, vertigo, and periods of excitement which may sometimes take the place of the convulsive attacks and present very similar characteristics to those met with in general epilepsy of sudden onset.

Ophthalmic migraine may be looked upon as a partial sensorial epilepsy and merits a brief description. It begins with a phase of excitement, violent pains, scintillating scotoma, etc.; then follow a phase of exhaustion, hemianopsia, and sometimes somnolence.

Ophthalmic migraine is characterized by various ocular disturbances (most frequently luminous sensations), together with a headache which, springing from a point limited to the temple, soon radiates over the entire half of the cranium corresponding to the affected eye. Then, as a rule, nausea and vomiting succeed. The migraine often occurs in the form of periodical attacks which are similar in the same patient but may vary in different persons. It may be associated with partial convulsive epilepsy or may alternate with it.

Nearly always the visual disturbances manifest themselves first. Sometimes it is a slight dimness of vision, sometimes a temporary hemianopsia—the patient is surprised at seeing only half of every object. In some cases absolute blindness occurs. But there is another special subjective symptom which may appear alone or may be associated with the other disturbances named, and that is scintillating scotoma.

In general the scotoma occupies the peripheral portions of the visual field, most frequently the external portion; it is rarely central. It may present itself perhaps in the form of a larger or smaller ball of fire, perhaps in the shape of a toothed wheel of red, white, or phosphorescent aspect, and in rapid vibratory motion or rotating around its centre. When the patients experience this symptom for the first time they cannot give an exact account of it; they merely perceive a dazzling comparable to that observed in trying to look at the sun, or perhaps they have a sensation of fireworks or of a sheaf of sparks. Later, however, when they can give a better account of what they experience their descriptions are almost identical. The luminous cogwheel enlarges, its centre becomes obscure; gradually it passes beyond the limits of the visual field above and below, and the patient sees only a portion of it in the form of a broken luminous

line which continues to vibrate until it has entirely disappeared. Occasionally the scotoma consists of a simple luminous zigzag resembling an electric spark. These luminous sensations persist when the eye is closed.

When an observer has been able to examine the eye at the moment of onset of the migraine he has found the pupils usually contracted, often unequally so, that of the affected side having been the smaller (Latham). Galezowski, however, in a case of migraine with periodical blindness, has observed dilatation of the pupil.

The visual disturbances are frequently accompanied by neuralgic pains or by a sensation of tension in the affected eye which is sensitive on pressure. Sometimes the visual disturbances and pains in the globe are so intense that the presence of acute glaucoma is suspected (Dianoux).

The headache, which appears at the end of some minutes, sometimes half an hour, an hour, and even longer, is located in the frontal, infraorbital, or perhaps the temporal region on the side of the affected eye. This pain, which the patients sometimes compare to a nail driven into their cranium, is often relieved by pressure. It soon radiates over the entire half of the cranium. The head is hot, the arteries beat strongly; the pain is increased by movements. Frequently, too, a sensation of vertigo which may be more or less pronounced is superadded to the cephalalgia, and the termination of the attack is marked by nausea generally followed by vomiting.

Aside from these complete attacks, there may be abortive forms of ophthalmic migraine. Thus, ocular disturbances recurring periodically may alone constitute the malady; some patients complain of sudden and transitory losses of vision (retinal epilepsy or epileptic amaurosis of Hughlings Jackson). In other instances, on the contrary, the visual disturbances are so slight that the patient's attention must be called to them in order to make him notice them. The nausea and vomiting at the end of the attack are not constant. It may happen that these migraines, while incomplete at first, may subsequently become complete. Sometimes the symptoms are disconnected, that is to say, the ocular disturbances and the migraine, for example, manifest themselves at intervals but separately.

Ophthalmic migraine is associated sometimes with partial epilepsy and also with ordinary epilepsy. What tends to establish still more firmly the relationship of this affection with epilepsy is the fact that it occasionally leaves behind not only a general weakness with somnolence, but true paralyses. In like manner we may observe hemianopsia persisting for a longer or a shorter time after scintillating scotoma; at other times the sequelæ are dulness of hearing, of

taste, or of smell. Even true transitory paralyses affecting the tongue or the arm have been noted as a result.

Like convulsive partial epilepsy, ophthalmic migraine may appear in serial form, recurring several days in succession. Sometimes the attacks are subintrant and terminate in a period of stupor. They then constitute what I have termed the status of migraine (*état de mal migraineux*[2]).

Ophthalmic migraine is, taken altogether, an accident of slight gravity when the subject is young; but when it occurs at the age when arteriosclerosis is frequent the temporary disturbances may become permanent, the paralyses persisting sometimes as monoplegias, sometimes as hemiplegias, occasionally even as diplegias. In one case I have seen a pseudobulbar diplegia terminate fatally.

Permanent aphonia is not a rare sequel of these migraines. Other associated sensorial phenomena may be produced by epileptiform migraine: hearing, smell, and taste may become affected with subjective sensations. Besides the epileptiform motor discharges, migraine is frequently accompanied by objective disturbances which are not without interest; there is some degree of spasm of the orbicularis and of the eyelids, the facial muscles of the painful side are stiff, the face is slightly distorted; the sternocleidomastoid is likewise contracted and draws the head away from the painful side. Sometimes the migraine is associated with local asphyxia of the extremities, which shows that there exists at the same time a spasm of the peripheral vessels.

Ophthalmic migraine is occasionally accompanied by motor impulses, constituting one of the most important characteristics which show the relationship of this affection to epilepsy.[3] Mingazzini[4] has recently described some mental disturbances in migraine which would argue in the same direction.

Sensory partial epilepsy may be more or less widely distributed over half the body; it manifests itself by crises of painful numbness or tingling (Charcot, Löwenfeld, Pitres). It is rather frequent in general paralytics.

Ophthalmic migraine may be compared with ophthalmoplegic migraine in which the paralysis affects the muscles of the eyelids or of the globe. Moreover, some facts are to be observed which establish the relationship of migraine to localized spasms of that region. These facts are not without analogy with the facial neuralgias described as epileptiform (Trousseau) and which really should be called epileptic as I have shown,[5] when we see that the spasm involves the muscles in closest proximity to the affected sensory nerve.

General Epilepsy.

Symptoms.

The epileptic attack may occur without prodromes and strike down a person in good health. But this is far from being always the case, and Lasègue's opinion, "that every person who has an aura is not an epileptic," is much too exclusive. The great majority of patients have premonitions of their attacks.

Premonitory Symptoms.

Sometimes we observe remote premonitory phenomena which may show themselves several days before the attack. Occasionally they are motor disturbances, such as tremors, general or local muscular twitchings, mumbling, grinding of the teeth, particularly at night; or else a kind of muscular slowness or awkwardness; or again some difficulty of speech. Sometimes the patient experiences various subjective sensations, buzzing in the ears, tickling of the throat or of the nasal mucosa causing coughing or sneezing. At other times it is a genital excitement, erections, nocturnal pollutions, or else visceral disturbances, impaired digestion, difficult respiration, or oppression. Not rarely hallucinations are observed; some epileptics perceive a pleasant or disagreeable odor which is always the same before each of their attacks; others have visual hallucinations. Some hear voices insulting them, making disagreeable remarks, etc.

These prodromes may also be limited to change in character; a certain patient becomes irascible and quarrelsome, another exhibits an exuberant gaiety. It is necessary to distinguish these remote precursory phenomena from the aura which immediately precedes the attack.

The aura is a peculiar sensation which extends from some part of the body or of the extremities towards the head. It is very frequent; O'Connor has found it in seventy-eight cases out of one hundred. It has been compared to a vapor, to which fact is due the name which it has borne for centuries (Galen). But it is far from assuming always the same form, and following Delasiauve we may divide the auras into motor, sensory, sensorial, and intellectual.

The *motor* aura manifests itself in the form of twitchings, tremors, or muscular palpitations which appear first at the periphery of a limb, the hand for instance, and successively extend to the forearm and arm; then the attack follows. Sometimes we observe a simple twitching of the eyelids or perhaps some mumbling. Some patients

are noticed to perform certain coördinated movements; they begin to walk or to run (aura cursativa), others execute a gyratory movement at the end of which they fall and lose consciousness. The epileptic attack is also sometimes preceded by yawning, to which may be added stretching of the arms, called pandiculation. Or else the onset of the attack is indicated by sneezing, hiccough, or a spasmodic cough.

The *sensory* aura consists of a sensation of hot or cold vapor which extends from a point on the extremities or the trunk. In other instances it is a numbness, formication, or some sort of a pain. Sometimes there is experienced a sensation as of a ball rising into the throat, which it may be difficul tto distinguish from globus hystericus.

The *sensorial* auras may affect all of the organs of special sense, especially sight, hearing, and smell. Thus there may be a retinal hyperæsthesia which renders daylight unbearable, or else a dimness of vision which may amount to absolute blindness. Certain patients have visions of plain colors among which red is most frequently mentioned, in others the colors or strange objects are such that no exact description can be given. More perfect visual hallucinations are also observed, such as sudden apparitions of known or imaginary persons. Megalopsia and micropsia should also be included in this group.

Auditory auras are equally frequent. They consist of a sudden reduction in the acuteness of hearing or perhaps the opposite condition, a hyperacuity which may be so great as to render the perception of the least noise painful. Sometimes the patient hears humming sounds, whistling, strange noises, or perhaps insulting words, voices that call him, and he falls at once.

When the attack is preceded by an olfactory aura the sensations described are nearly always disagreeable (rotten eggs, carrion, etc.); more rarely the odors are pleasant.

The gustatory auras, finally, which are infrequent, consist most often of an unpleasantly bitter, metallic, or nauseating taste.

The viscera, too, are sometimes the seat of varied sensations which immediately precede the attack; thus the onset may be marked by violent palpitation of the heart or by a more or less intense precordial pain. In certain cases it is a painful oppression or perhaps a laryngeal spasm with a sense of constriction of the throat. The aura may also spring from the abdomen and be characterized by a sense of weight or painful dragging at the stomach, together with regurgitations, nausea, and even vomiting. Sometimes it consists of violent colic, rectal or vesical tenesmus, or an urgent desire to defecate.

Finally, we observe *psychical* auras which may assume all possi-

ble forms. Sometimes the aura consists of great excitement: one patient suddenly becomes quarrelsome or furious, another is seized with an irresistible impulse, he runs away and commits odd or improper acts which terminate in the attack. Sometimes it is a state of depression and melancholy with enfeeblement of intellect, loss of memory, etc. Or again an involuntary terror seizes the patient, a sensation of danger or of imminent death, or the fear that he may kill himself without any suicidal impulse; or perhaps he experiences an unexpected change in his natural affections. Mention may also be made of the dreamy state with reminiscences (Jackson), etc. Nocturnal attacks are often preceded by dreams.

All these premonitory phenomena are far from presenting themselves in an isolated form in each patient. Frequently they are variously combined; thus it is not rare to see a sensory aura accompanied by muscular disturbances in the extremity affected by it, just as a visual aura may be associated with prickling of the conjunctiva, or an auditory aura with a sense of contraction of the facial muscles. It is safe to say, however, that an aura is almost uniformly the same in each patient and that it frequently constitutes a significant indication to the sufferer and those about him. Pretty often, too, the epileptic aura remains for a long time in an isolated condition before it is accompanied by convulsive disturbances.

During the phenomena of the aura we can demonstrate in the patients a slight elevation of the general temperature which increases to the onset of the convulsive attack. Even a local hyperthermia has been noted in the limbs which are the seat of the aura. In like manner it is possible to prove the existence of an increased arterial pressure by means of the sphygmometer.

Thorion[7] claims that the epileptic crisis is preceded by a greatly increased elimination of the urinary elements, a veritable discharge; this chemical aura, he thinks, might even enable one to predict the return of the attack.

Symptoms of the Attack.

Classical authors describe under the name of *grand mal* the convulsive attacks of epilepsy and reserve the name of *petit mal* for the vertigo and mental hebetude. I think it incumbent upon me to warn the reader at the beginning of this study against the too schematic character of the usual descriptions. While these permit of a classification, which it is convenient to present and easy to remember, of the different symptoms, it must be with the reservation that they cannot be applied to any one case in particular. In a word, there are almost as many forms of attack as there are epileptics.

The *major attack* may be divided into three stages: the tonic stage or that of tetanic convulsions; the clonic stage or that of irregular convulsions; and the stage of stertor.

Whether or not the attack is preceded by precursory phenomena or an aura, it is generally of sudden onset. The patient grows pale, emits a cry, and falls unconscious. All the muscles of the body are in a state of tetanic rigidity. The eyes move upwards and the lids close convulsively; the pupils are widely dilated and insensible to light. The contracted neck muscles force the head backwards or sideways; the features are drawn, the jaws are set, and the tongue is often severely bitten; the limbs, extended and rigid, are agitated by extremely rapid small muscular contractions, a veritable vibration, of which we can convince ourselves by the application of the hand. At the same time the movement of the thorax is arrested in a prolonged expiration, and the face which at the moment of the fall was pale becomes congested, red or purple. The circulation is so greatly impeded that the vessels often rupture and one may notice as a consequence the occurrence of ecchymoses of the lids, for example, hemorrhages from the nose, ears, bronchi, or even into the cerebrum. The abdominal muscles are likewise made tense during the tonic stage, and often urine and fæces are expelled.

The entire stage lasts but a few seconds. The general tension of the muscles then diminishes gradually and the stage of clonic convulsions commences. During the tonic stage the pulse is strong and frequent, the arterial tension is greatly increased. In some cases it has been possible to examine the fundus of the eye and to demonstrate a narrowing of the retinal arteries which soon gives way to an intense venous congestion.

After the lapse of some seconds the whole body becomes agitated. The head is violently drawn back or perhaps executes rapid rotary movements; the eyelids twitch, the eyes roll in their sockets, though remaining convulsively turned up so that nearly the entire cornea is hidden by the upper lid. The muscles of the face are in incessant motion, giving to the features a grimacing expression which is hideous to the onlooker; the jaws open and close, the teeth grind, the tongue projecting beyond the mouth is bitten and raw. At the same time the respiratory movements are hurried and jerky; a bloody foam escapes from the mouth, and a mucous discharge flows from the nose. The respiration is rattling; sometimes the patient actually roars. The limbs, shaking violently, are agitated by the most various contortions. As a rule, the thumb is forcibly flexed into the palm of the hand, covered by the other fingers; but this attitude is not absolutely constant, any more than it is peculiar to epileptic convulsions, as

has been claimed. Nearly always the convulsions predominate on one side of the body.

During the clonic stage the arterial tension remains elevated, the pulse is frequent, the heart beats are tumultuous and even more accelerated than in the preceding stage. The urine may escape at this moment, simply owing to the relaxation of the sphincters; the contractions of the intestines and of the abdominal muscles cause borborygmi. During the entire attack the loss of consciousness is generally absolute and the patient is insensible to all irritations. Nevertheless, this loss of consciousness, which until recently has been looked upon as an essential characteristic, may be lacking even in convulsive attacks which are regular in every other respect.

After several minutes the convulsions become less extensive and less frequent, and little by little they cease. Respiration becomes regular, deep, and associated with sonorous rhonchi; this is the commencing stage of stertor. The patient then is in a state of profound relaxation: the limbs are flaccid and inert, the tendon reflexes are often abolished; the insensibility is as complete as during the onset. Gradually the face, which was red or purple, grows pale and assumes a livid, cadaveric hue. Respiration is noisy; the mouth, filled with thick saliva, exhales a repulsive odor. A transudation, often fetid, appears on the body and face.

The stage of stertor lasts from ten minutes to a half-hour; rarely it is prolonged to an hour or two. Then the patient opens his eyes, casts a dull look about him, lifts his head, and rises, having as a rule no recollection of what has passed. For the rest of the day he remains in a state of somnolence and general lassitude, which is sometimes accompanied by a feeling of soreness in the limbs.

Such is the classical and complete attack of epilepsy; but it does not always present itself in this immutable form. The succession of tonic and clonic stages is not of invariable occurrence, as has been stated above. Either one may be absent, or perhaps the clonic stage may appear first. Sometimes the attack consists of a simple rigidity without apparent movements, or is perhaps associated solely with some tremors. The convulsions may fail to occur in one or another portion of the body, in one extremity, in the face, in both lower extremities, etc.

When the attack occurs at night, it may pass unperceived unless the patient is awakened by the prodromal phenomena; the stertor then merges into the normal sleep without interruption. Epileptics have been observed who, having only nocturnal attacks, were unconscious of their disease for years. Often, however, the patient is made aware of it by the evacuation of urine, by the bitten tongue discovered

on awaking, by an unaccountable lassitude, or by the presence of subcutaneous ecchymoses.

The major convulsive attack with loss of consciousness presents this constant characteristic, that it leaves no trace in the memory of the patient. This amnesia, however, may show considerable variations as to its duration. Many patients remember the remote prodromal phenomena and even the sensations of the aura. Some retain the memory of the first convulsive movements and there are those who hear their cry. Rarely consciousness is completely retained. Sometimes, on the other hand, a retroactive amnesia exists, which comprises the facts which have preceded the attack for a longer or shorter time.

After the attack mental retentive power is abolished or diminished, in like manner as the receptivity during a variable time after apparent awakening: the patient perceives and retains recent impressions badly.

The epileptic attack does not always occur in the same manner. In one and the same patient it may vary infinitely in its aspect, its intensity, and its duration. Moreover, along with the classical description of the major attack attenuated types have been dwelt upon, which have been designated by the names of vacuity, vertigo, incomplete attacks, etc.

The vacuity is limited to a loss of consciousness, with temporary pallor of the face. The patient does not fall or utter a cry, but he remains motionless, interrupting the act in which he is engaged or the conversation he is holding. He continues for a moment in that condition, with fixed look and without any convulsive movement. Sometimes he mutters automatically a few incomprehensible words (muttering epilepsy). After a few seconds he regains control of his senses, often without knowing what has happened to him. The vacuity generally leaves behind some dulness of the intellect, a state of more or less marked obtuseness.

In vertigo the patient falls as a rule and suffers a temporary loss of consciousness; but the convulsions are very slight and may pass unperceived. The stage of stertor is much shorter if it exists at all.

By the term *incomplete attacks*, which must not be confounded with attacks of partial epilepsy, paroxysms are designated in which one or more of the most important phenomena are absent, such as the initial cry, biting of the tongue, and involuntary micturition, or in which the convulsive phenomena are limited to certain groups of muscles. For instance, the convulsions may be limited to a few rotary movements of the head, some grimaces of the face, or perhaps a few twitchings of the upper extremities. In other cases there may

be only some sensorial phenomena, such as auditory and gustatory sensations, peculiar visceral sensations of strangulation, thoracic or abdominal constriction, or else some secretory disturbances like sudden salivation, etc. The patient may not fall, the loss of consciousness may be very short or absent. The stage of stertor and subsequent hebetude are much less marked.

Herpin maintains that the existence of incomplete attacks before the appearance of the convulsive crises is almost the rule, and he has endeavored furthermore to prove that the incomplete attacks reproduce in general the premonitory disturbances, the prodromes or aura; and finally that when the major attacks are recovered from they become progressively attenuated and transformed into incomplete attacks.

Sometimes the attack takes the form of shocks or tremors, which have been justly considered as aborted manifestations of the disease. In this class belongs the salutatory spasm (spasmus nutans) which is observed mainly in children. The convulsion consists in a salutation, a forward inclination of the head with slight bending of the trunk, which is repeated twenty, thirty, or one hundred times a minute. There are attacks which are constituted solely by this salutation, and which may recur several times hourly. In some persons this is associated with a brief pallor of the face, a fixed look, and dilatation of the pupil; in others there is even loss of consciousness which lasts as long as the attack. The convulsions may present some variations; thus the head alone may take part in the movement, or else the shoulders are lifted simultaneously and the arms are agitated by muscular spasms. In other instances the nodding, instead of being in an antero-posterior direction, is executed obliquely in consequence of an isolated contraction of one sternomastoid.

Children suffering from this affection may recover, but sometimes they present later on major attacks of ordinary epilepsy.

It may happen that the convulsive manifestation is limited to muscular contractions affecting either a limb or the facial muscles, thus assuming the appearance of a true tic, or perhaps the spasms may be general. These convulsive movements, which occur in general in confirmed epileptics, in the intervals between attacks, often appear after longer intermissions in serial crises which may last several hours or even days. They result spontaneously, or are produced by an accidental cause. When they are repeated for a long time, they may be followed by symptoms of weakness and of intellectual impairment, like ordinary attacks. They may constitute a veritable status epilepticus lasting several days.

In certain cases the convulsive phase of the major attack is en-

tirely absent, and there is nothing but an ictus apoplectiformis, followed by stertor. Trousseau lays particular stress upon this form, which it is sometimes difficult to recognize as a manifestation of the disease, especially when it occurs in a person not known to be epileptic or to have suffered from the ordinary major attacks.

We must recall the resemblance to apoplexy of the paralytic attacks, which often assume the paraplegic form: the patient sinks down without losing consciousness and rises at once.

Still more abnormal manifestations of epilepsy have been described, such as sleeping crises preceded or not by an imperative and sudden desire to sleep. A more or less pronounced stertorous state constitutes the whole attack. Attacks of narcoleptic sleep have been observed by Weiss, Westphal, Fischer, and others; like other epileptic manifestations this sleep may yield under the influence of the bromides. Kesteven has cited an instance and I have seen another.* In some patients the major convulsive crises are replaced by fits of trembling, which may last several hours.*

Another form of epilepsy, known from antiquity, is described under the name of procursive epilepsy, because the patient begins to march or run like an automaton. Sometimes preceded by an aura and accompanied by a cry, the course is in general an unconscious one. It may constitute the whole of the attack or may precede it; the patient, having rushed forward, stops suddenly and falls. In other cases it forms only a concomitant feature of the convulsive attack; the patient rises and starts to run before he regains consciousness. This form of the disease belongs chiefly to infantile epilepsy. While it is often isolated at first, it alternates later in the adult with convulsive seizures, or gives place to them.

The flight of epileptics may be considered as a variety of this form: the patient hastily leaves his domicile and commits acts which are often strange and incoherent, but sometimes also coördinated and complex, such as buying a railroad ticket, embarking, etc. The amnesia of all the acts performed may be complete. I shall consider later at greater length these acts of ambulatory automatism, in connection with the mental disturbances of epileptics.

Duration of the Attack.

The epileptic fit lasts barely more than two or three minutes. Lasègue went so far as to claim that its duration never exceeds a minute, an opinion which is not tenable. We may observe attacks which last even five, eight, or ten minutes; still the brief duration of the convulsive seizure is an important element of differential diag-

nosis from hysterical attacks for instance. The relative duration of the stages which compose the attack is likewise variable; it may be said, however, that the tonic stage is generally the shortest. Sometimes, it is true, the stage of stertor is the shortest; but as the stertor often passes without transition into the sleep which follows the attack, the duration of this stage is not always easily estimated.

Frequency of the Attacks.

The frequency of the fits is extremely variable. There are patients who have but a single attack in their lives. Others are free for several years after having had numerous fits. It is not rare to see patients who have one or two attacks in the year, or the fits may recur once a month. In women the coincidence of the attacks with the menstrual periods has been known for a long time. In the gravest forms of the disease the attacks occur weekly, or several times each week. Epileptics who have one or more attacks every day are patients requiring continual surveillance; their intelligence is usually more or less impaired.

Trousseau has emphasized the fact that epileptic fits often occur at night, especially in the beginning, so that they may for a long time pass unperceived. In fact, the statistics give a large number of nocturnal attacks. Some have thought to see therein the influence of the horizontal position; still an almost vertical position during sleep has not exerted any preventive power. It is more likely that dreams and nightmares have some influence upon the nocturnal return of the fits, and sometimes it has been possible to prevent them by keeping the patients awake. There are patients who have their attacks solely at night, others have them more frequently during the day.

Nearly all epileptics who have major attacks present at one time or another incomplete seizures in the shape of vertigo or simple vacuity. In some the disease is even limited to such abortive manifestations; these are said to suffer from *petit mal.*

The great majority of patients present occasionally recrudescences in the number of attacks, either without appreciable cause or under the influence of some causative factor. The menstrual period has been mentioned above; excesses, fatigue of any kind, and moral emotions may act in the same way.

At all times the influence of intercurrent diseases on the course of epilepsy has been pointed out. A certain number of febrile affections result in a temporary suspension of the fits; among these are typhoid fever, small-pox, erysipelas, measles, and prolonged suppuration. Sometimes the improvement persists even after the cure of the inter-

current disease. In some fortunate cases a complete cessation of the attacks has been observed. But it would be erroneous to believe that certain infections could be utilized for the cure of epilepsy; in many cases they aggravate it.

The attacks may succeed each other in series, with more or less long intervals of respite; thus we may see patients who have twenty or thirty fits in succession in a single day. The number may be still higher. The serial attacks are associated with an elevation of temperature which is much greater than in isolated paroxysms and they are followed by a much more profound depression. But one important distinction is to be observed, namely, whether the patient regains consciousness between the attacks, or whether a fresh attack comes on before the preceding one has terminated. In the latter case the attacks are subintrant, or imbricated, to use Trousseau's expression. To this latter form the term status epilepticus has been applied, and it constitutes a veritable acute phase of epilepsy.

The status epilepticus, which was known to the older writers, has been particularly closely studied in recent years (Browne, Bourneville, and others). It may set in suddenly; sometimes it is preceded by several ordinary convulsive crises. It is characterized by an almost incessant repetition of the spasmodic convulsions. Sometimes a short interval separates the paroxysms, which are complete in their ordinary phases, and the attacks can be counted. Their number has been known to rise to one hundred and more in the twenty-four hours. But the characteristic point of the status epilepticus is that the patient does not regain consciousness between the attacks. The condition is always very serious: the temperature rises to 40° or 41° C. (104° or 105.8° F.); the pulse becomes extremely rapid, respiration is also accelerated, and death may ensue in the midst of the convulsions. As a rule, however, the convulsions become less intense after some time; the patient remains in a condition of stertor, with limbs completely relaxed, and is insensible to irritations; the pupils continue widely dilated. During this time the temperature remains high or perhaps even rises still more and death follows. The status epilepticus generally lasts two or three days. It is known, however, to have been prolonged to eight or nine days.

The prognosis is not absolutely fatal and the patients may recover. The fits then become less frequent and less violent, and the temperature falls. Consciousness returns little by little, but the patient is always very slow to rise. This is because the weakness is extreme; the stupor takes a long time before it is dissipated. The loss of weight, the diminution in the proportion of hæmoglobin in the blood,

and all the symptoms of debility which regularly follow the attacks and which will be discussed later, have reached the highest degree. The mental condition may be very seriously affected.

One fact to be noted is that the status epilepticus is not necessarily constituted by major attacks, but may result from a succession of incomplete seizures, vertigos, and fits of partial epilepsy. Under the title of the subintrant syncopal state in a nursing infant, Dauchez has described disturbances accompanied by convulsions which are manifestly of an epileptic nature.[10]

Under the title of epileptic equivalents or succedanea have been described a certain number of sensory, sensorial, and psychical symptoms whose connection with epilepsy is demonstrated by their coexistence in the same person or in the patient's family, by their essentially paroxysmal course, and by the weakness which they leave behind. Quite a number of them may be considered as incomplete seizures, limited, for example, to painful or convulsive manifestations of the aura. Some of them present a symptomatic character which is identical with that of certain organic affections, and may lead to mistakes unless account is taken of the conditions under which they are produced.

Thus among the visceral equivalents of epilepsy figures angina pectoris. Sometimes it follows, sometimes it precedes, and sometimes it coincides with epilepsy in the same patient. Moreover, the convulsive seizure may set in with a violent pain starting in the hand and extending to the neck and the precordial region. In that event, it is impossible to mistake the localizations of angina pectoris. The entire attack may be limited to this painful aura.

The attempt has been made to connect with epilepsy crises of palpitations and paroxysmal tachycardia; but to render this interpretation plausible it would be necessary at least that the patients presenting these abnormal crises should have some disturbance which belongs more manifestly to the epileptic series.

The same is true of certain forms of asthma which unexpectedly affect persons in good health who are free from any pulmonary trouble. The coexistence of asthma with psychical disturbances such as hallucinations, terrors, and fear of death is, besides, a well-known fact at the present time, and establishes an additional analogy between this neurosis and epilepsy. In young children who very rarely suffer from asthma, the affection may be observed to alternate with more or less generalized convulsions.

It is likewise correct to include spasm of the glottis among the incomplete seizures. Valleix and Trousseau have already described the relation of this malady to infantile eclampsia. The symptomatic

analogy with this affection is very great, a fact of which we may convince ourselves by the description given by Rilliet and Barthez of spasm of the glottis: "The infant is seized, suddenly and without prodromal symptoms, with an attack of convulsive suffocation. Respiration is suspended, the face is darkened and injected, the head is drawn backwards, the mouth is wide open; the child is restless and carries its hands to its neck as if to remove the obstacle to respiration; then, after a few seconds of this dyspnœa, it makes several short inspirations—whistling, sharp, and jerky—without intervening expirations, soon followed by a faint and wailing inspiration, or sometimes by a convulsive, noisy, and jerky expiration. At the same time the extremities stiffen, the thumb is carried into the palm of the hand, the fingers being stretched or flexed on the metacarpus . . . involuntary evacuations take place. Most frequently consciousness is preserved, sometimes it is abolished." In one case of spasm of the glottis observed in an adult, Brodher referred to the loss of consciousness and the consecutive drowsiness.

False croup is a convulsive manifestation to be grouped with asthma and spasm of the glottis as regards its relationship to epilepsy.

Among the visceral equivalents of epilepsy mention should be made of gastric disturbances, enteralgias, gastralgias, vomiting, and nausea occurring suddenly and without known cause in a person free from disease of the digestive apparatus. Trousseau has called attention to nocturnal incontinence of urine as a premonitory sign of epilepsy. Often it is merely a symptom which reveals an attack which has passed unperceived.

Epileptics are also subject to various painful phenomena which may be considered as equivalents of the paroxysm. Such are the migraines. Moreau de Tours remarked that the migraines of epileptics leave behind a profound stupor. They often develop and disappear with the same suddenness as the epileptic paroxysms. Besides, the connection between them has been admitted by some authors (Tissot, Parrot, Lieving); and in some patients a periodical migraine has been observed to give place to epileptic seizures which recurred at similar intervals.

All the above-named sensorial phenomena may take place without consecutive seizure. Thus on questioning the patients minutely we may trace in the intervals of the attacks certain sensorial perversions which may consist of visual disturbances, such as clouds passing before the eye, which the patients call dazzling, or else various hallucinations. Some patient suddenly sees objects become immeasurably large, another hears noises or voices which make him turn his

head, or else he perceives a peculiar odor or a disagreeable taste, which disappears in the same way.

These are the sensorial forms of epilepsy which have been studied in recent years especially by English and American authors (Hammond, McLane Hamilton and Anderson); there is no doubt that all these paroxysmal phenomena are connected with the cause, whatever it may be, of the central irritation that gives rise to the major convulsive attacks.

Can we admit the existence of dystrophic paroxysms? Young looks upon epilepsy as causing rapid emaciation and temporary alopecia; but in order that the proof be furnished it should be shown at least that these phenomena are observed in epileptics and apart from the periods of debility following the paroxysms in which they have a special significance. Still I have observed in confirmed epileptics variations in weight which appeared to have no connection with the frequency of the paroxysms."

It is the psychical forms of epileptic equivalents which are much more important, and to which we shall devote more attention. In speaking of the incomplete or aborted manifestations of epilepsy, I have described the mental vacuity which is in reality merely a momentary suspension of the psychical functions without apparent convulsions. But beside this form of vacuity with complete inactivity there is a dulness associated with impulses, that terrible manifestation of epilepsy which makes the epileptic sometimes dangerous to his family and to society. We are indebted chiefly to Morel for the description of these forms of masked epilepsy.

The paroxysmal mental disturbances which will be discussed may precede or follow a convulsive attack that may be of the major form or an incomplete seizure, or perhaps a simple vertigo; but to-day it is admitted without question that they may occur independent of any paroxyms of this kind, Legrand du Saulle to the contrary notwithstanding. They then constitute a veritable psychical equivalent of the attack, and with Maudsley we may compare them to a mental convulsion.

These mental disturbances may manifest themselves in the form of strange and violent impulses of short duration. Some patients suddenly begin to utter incoherent words, to exhibit rude behavior, and to injure persons about them. Others commit acts of brutality, strike people, and break objects. Under the influence of impulsion an epileptic is capable of wounding or killing persons who approach him, and it is even more terrible that his strength at such times is prodigious and that he strikes with remarkable precision. Some patients commit indecent acts; among the insane who exhibit their

genital organs in public Lasègue has shown that a considerable number are epileptics. After a few moments the patient recovers consciousness without retaining a single recollection of what has occurred, and he falls into a state of profound prostration unless a fit terminates the scene.

The vacuity associated with impulsive acts is not always of so short a duration. It may last several hours, and has been known to be prolonged for several days or a whole week (Charcot). The epileptic then is able to commit crimes necessitating the performance of complicated acts (theft, incendiarism, etc.). However, the acts thus performed are sometimes very reasonable in appearance, and do not attract attention. A patient may leave his house, walk straight ahead, or even take a journey without anybody seeing anything abnormal in his behavior. Corroborative of this is the case related by Lasègue and Legrand du Saulle of a patient who took passage at Havre and did not come to himself until he was off Bombay. It should be added that, when they are in this mental state, the patients are often subject to various and sometimes terrifying hallucinations which may play an important part in determining their impulsive acts.

What characterizes the impulsive acts of epileptics is their suddenness and their apparent independence of external circumstances. A peculiar psychical state often precedes them: a restlessness, an abnormal irritability, sometimes a true sensorial or other aura. Another characteristic is their recurrence in the same form in the same person; for instance, one patient will regularly present a seizure of pyromania, and it will be noted that he sets the fire always in the same manner. In the case of acts of violence the force displayed is always out of proportion to the object to be attained. If it be a murder, the victim will be covered all over with wounds and the epileptic frequently throws himself fiercely upon the cadaver.

Finally, the paroxysm ceases as suddenly as it began and is often followed by a state of depression, a debility similar to the one which succeeds the convulsive crises.

As a rule, the epileptic does not retain a single recollection of the acts performed during the impulsive crisis, and it is admitted that unconsciousness is a fundamental characteristic of epileptic delirium. The question is a rather difficult one for solution, for it will be readily conceded that it is not sufficient for an act to leave no trace in the memory to enable us to say that it is accomplished without participation of consciousness. Thus when we see an epileptic, under an impulsive influence, perform complicated acts which require the operation of his cerebral functions, when we see him conduct himself in

harmony with the conditions and the circumstances in which he finds himself, it is not unreasonable to suppose that he is conscious of his acts at the moment of their performance. In reality the epileptic finds himself in the same psychical conditions as a person acting under the impulse of some passion. The amnesia which follows these psychical discharges is in every way comparable to that after violent emotions or even to traumatic shocks which determine a nervous exhaustion with retroactive amnesia.

It happens, however, sometimes that the patient is fully conscious of the acts he commits and remembers them. The impulse to their commission is none the less irresistible, and is associated with acute mental suffering. Falret has described these forms of impulsive delirium under the name of intellectual *petit mal.*

In other cases the patient, recovering his reason suddenly in the course of his delirium, finds himself with the instrument of the crime in his hand, or surrounded by such incriminating evidence that he cannot for an instant doubt that he is the author of the act. Under such circumstances the patients have been seen to be humiliated and to seek for some semblance of reason for their act, recognizing that it has been accomplished in full consciousness of the cause which they connect even with a fact in their previous lives. It is evident how great an interest attaches to the discovery of the truth from a medico-legal standpoint in a similar case.

The psychical paroxysm may also present itself in the form of acute mania, whose gravity varies from simple excitement with incoherent loquacity, hallucination, and insomnia to furious mania described by Falret under the name of intellectual *grand mal.* These major seizures of furious mania (acute delirium of epileptics, of Cossy) may break out suddenly or after some prodromes resembling those preceding *petit mal.* Sometimes they manifest themselves after a seizure or after repeated attacks of vertigo. The patients become irritable and overwhelmed by furious fits of passion against objects and persons. The talk incessantly, and give vent to the grossest insults. The features assume an expression of ferocity mixed with terror or suffering; the eyes sparkle, the congested face is covered with sweat under the influence of alarming hallucinations; the patients give way to a perhaps unheard-of violence and are so much the more terrible since they are insensible to the severest pain and since their movements are often remarkably sure.

The seizure rarely lasts longer than some hours when it attains this intensity; but when it is repeated the general condition quickly becomes exceedingly grave. The tongue becomes dry, the voice hoarse and finally inaudible. The temperature rises; it has been ob-

served as high as 39° to 40° C. (102.2° to 104° F.) when the maniacal attack lasts several days, which is rare. The patient then falls into a state of profound depression similar to that following the convulsive status epilepticus. Death has been seen to occur under such circumstances. On the other hand, if the maniacal attack is of short duration the return of reason is rather sudden and the patient complains only of headache or a general weakness, retaining merely a vague recollection of what has occurred.

Epileptics sometimes have fits of depression during which they are indifferent to everything, although they manifest no delirium; these seizures, which display the characteristics of mental confusion, have seemed to me to deserve the name of fits of apathy."

Besides these paroxysmal intellectual crises we occasionally observe more prolonged forms of insanity alternating with periods in which mental soundness is interrupted by epileptic attacks, which are completely lacking while the mind is affected. Haslam has described instances of this nature.

These varied manifestations of epilepsy present a twofold common characteristic, namely, they constitute, first, phenomena of excitement in the shape of a veritable nervous discharge, and, second, they leave behind them a state of exhaustion corresponding to the intensity of this discharge. These symptoms of exhaustion occupy a prominent place among the sequelæ of the paroxysms, and I have made a study of a number of them. I am convinced that they never fail to occur; and when the excitement persists after the convulsive phenomena, when the arterial tension remains high, when the physical activity continues exalted and the muscular power exaggerated, it is safe to say that the nervous discharge is still incomplete and that the depression will ensue at a given moment.

The nervous exhaustion following the attacks manifests itself not alone by disturbances of motility, sensibility, and intelligence, but also by disorders of general nutrition. It may be said that it is not a particular organ or function which is altered, but that the entire machine is worn out.

The weakening of the muscular power succeeding the convulsive attack is proportionate to the gravity of the paroxysm, but it may be said to be constant, since we may convince ourselves by dynamometric and dynamographic examinations of the manual pressure exercised after the nervous discharge and in the normal state; the pressure is not only feeble, but it is slow. Sometimes it is found equal on both sides, but more frequently it is more powerful on one side; this fact corresponds with the usual unilateral predominance of the convulsions. The feebleness may also affect the lower extremities;

the gait then is hesitating or the erect posture may even be impossible.

Sometimes the muscular weakness manifests itself by a trembling of the limbs which lasts a certain period after the seizure. Reflex excitability of the cord is often diminished; the tendon reflexes then are feeble or even abolished immediately after the attack. Without speaking of the general relaxation which follows on the major attacks, we sometimes observe the occurrence of true paralyses of the extremities which may take the hemiplegic, paraplegic, or other form. The paralysis is apt to affect by preference the muscles in which the spasm predominated. It may be added, however, that these paralytic disturbances are generally very evanescent and that they are undoubtedly less frequent in ordinary epilepsy than after attacks of partial epilepsy. In some cases the paralysis is greatly limited, affecting perhaps the muscles of one-half of the face or of the tongue, or else it causes a temporary strabismus. I shall describe in passing the conjugate deviation of the eyes and of the face mentioned by Beevor as a sequel of epileptic attacks. It generally occurs on the side opposite to that on which the convulsions predominated.

Nystagmus following attacks, which I have been able to observe more rarely, is a phenomenon related to the paralytic trembling of the muscles described above. The nystagmus is sometimes associated with a temporary strabismus. Lateral nystagmus becomes exaggerated when the eye is moved to its extreme arc of excursion.

After intense nervous discharges it is often possible to produce, by percussion of the pectoralis major or pinching of the biceps, the phenomenon known by the name of idiomuscular contraction, whose presence again shows the exhaustion of the entire motor apparatus, for this condition seems to belong to adynamic states.

Transitory disturbances of speech may also appear as an immediate consequence of an epileptic fit. Sometimes it is merely an embarrassment, a hesitation in the pronunciation of words, accompanied by trembling of the tongue and lips resembling the speech disturbances in general paretics. Sometimes the disorder of articulate language is complicated with disturbances in the use of signs; the patients are unable to read. Or else it may be a temporary verbal deafness—they fail to understand what is said to them despite the evident attention they pay.

The post-epileptic exhaustion manifests itself also by various disorders of sensibility. Thus the complete anæsthesia of the stupor may not disappear at the same time from the entire surface of the body, and hence we sometimes observe after the seizure a more or

less pronounced anæsthesia affecting one extremity or perhaps a larger portion of the body.

In the special-sense organs, especially vision and audition, symptoms of deficiency are likewise to be observed. The most varied disturbances of vision have been described, such as diminution of visual acuity, amblyopia, concentric limitation of the field of vision, defective color perception (dyschromatopsia), abolition of some or all color perception (achromatopsia).

Post-epileptic deafness has been described (Russell, Bennett, Charpentier); impaired hearing is very common after the seizure, as are defective taste and smell. One remarkable fact, which is to be compared with similar observations in hysteria or with the sequel of certain cerebral lesions, is the possible coexistence of sensory and sensorial anæsthesia in the affected organs; for instance, deafness and anæsthesia of the auricle, amblyopia and insensibility of the lid, of the eyeball, and of the rim of the orbit.

On measuring the time of reaction, which can readily be done by means of d'Arsonval's chronometer, it is seen that epileptics react less quickly to irritations after a seizure than they do in their normal state. The prolongation of the time of reaction is a very natural consequence of the weakened sensibility and motility. It is likewise a phenomenon of stupor, the same in kind as the weakening of the memory and intelligence, which can be demonstrated in patients under similar conditions.

The time of association is also notably augmented during the post-paroxysmal period, and I have shown elsewhere that the association is frequently disturbed.[13]

These defects in psychical activity coincide with a loss of memory relating to circumstances preceding the attack. This retroactive amnesia may include events occurring several hours before the seizure.

The functions of nutrition, too, suffer by the epileptic paroxysm. Loss of weight is evident, especially after serial attacks. The elimination of phosphoric acid and nitrogen in the urine is increased. Albumin in the urine is rather frequent.

Subsequent to the attack certain drugs are eliminated more rapidly by the kidneys. The perspiration caused by pilocarpine is diminished and its onset is delayed. The skin and the hair remain dry. Sometimes the hair falls in patches; this post-epileptic alopecia, so far as I have been able to judge, is recovered from spontaneously.[14] After severe or repeated attacks the nails sometimes show transverse furrows. The lacteal secretion is often suspended and may be permanently suppressed by an epileptic seizure.[15] During

the epileptic fit the arterial tension remains high; if a sphygmographic tracing be taken as soon as the convulsions have ceased, as after every muscular effort, we find a rounded curve indicating a high tension; but soon the tension falls below the normal and may remain so for several hours. During the post-paroxysmal period the number of red corpuscles in the capillary vessels is diminished and they undergo a more rapid alteration, while the hæmoglobin is reduced; but in spite of the reduction in the number of corpuscles and in the amount of hæmoglobin the density of the blood is increased (Johnson Smith).

The post-epileptic impairment of nutrition manifests itself sometimes by the rapid formation of sloughs, by a delay in the cicatrization of wounds, by a special tendency to solar erythema, and by an accelerated progress of tuberculosis.[18]

Among all these sequelæ connected with the nervous exhaustion some generally disappear after several hours; these are the anæsthesias, the sensorial disturbances, and the paralyses. Others, however, persist much longer; sometimes the epileptic requires several days to recover completely from the crisis, especially to repair the loss of hæmoglobin and to regain his mental activity. It is not surprising, therefore, that the recurrence of the attacks leads in the end to a deterioration of the entire organism; the intellect finally perishes completely under such circumstances, and when we come to treat of the mental state of epileptics we shall see what importance is to be attached to the post-epileptic exhaustion in interpreting the forms of dementia which affect some of the patients.

It is safe to say that we very rarely meet with epileptics who are well balanced morally and intellectually. Their character is usually mobile, and traits of depression predominate. Habitually gloomy, irritable, and jealous, they may be observed to pass suddenly to sentiments of generosity, benevolence, and enthusiasm which vanish as quickly as the paroxysms to which they are subject.

The state of profound depression which follows the convulsive discharges presents a most favorable field for the development of feelings of sadness, inferiority, and impotence, under the influence of which suicidal impulses sometimes arise. Their frequent hallucinations, the terrifying dreams to which they are very liable, render such persons timorous, suspicious, and distrustful. Their tendencies often take an impulsive character; but rarely they may manifest tenacity of purpose or true energy. The mental oscillations which are so frequent among them have given rise to the view that remittent insanity and circular insanity are manifestations of masked epilepsy (Morel, Doutrebente, Soukey).

Even in those who enjoy the apparent possession of all their mental faculties memory is often feeble, comprehension difficult, and conception slow. This intellectual weakness increases after the paroxysms and is still more pronounced when these become more frequent and more intense. Side by side with these deficiencies we may sometimes observe sudden lighting up of the intelligence in the form of hyperamnesia or hyperideation, which occasionally occur as precursory signs of attacks, and which may last several hours and even several days. Nearly all patients of this class exhibit mental disturbances. As a general rule, these disturbances are more pronounced and lasting in proportion as the paroxysms are more frequent and more severe. Under such circumstances the patients may reach the borderland of absolute dementia.

It has been observed that epileptic dementia is more frequently the result of attenuated paroxysms (vertigo, absence, or muscular twitchings). This is because the latter occur in greater frequency, for it is beyond question that the major attacks leave behind a more marked exhaustion than the incomplete seizures, and when they recur equally often they result even more rapidly in dementia.

Epileptic dementia is a kind of stupor affecting the majority of the nervous functions. Delasiauve has described it very well in the following words: "According to the gravity of this state the attention is weak, inefficacious, or nil; memory is confused, treacherous, or entirely lost; judgment is uncertain, defective, or abolished; the conceptions are obscure, aborted, or false; the concatenation of actions is burdensome, incorrect, or impossible; the imagination does not soar; the resolutions are inert, fugacious, or without motive; the desires are beyond attainment and baseless. From this intellectual mutilation results as a necessary consequence the annihilation of moral, sentimental, affectionate, and instinctive manifestations." All other conditions being equal, dementia seems to occur more rapidly in proportion to the age of the subject.

One special character of epileptic dementia is its intermittent progress. It becomes more pronounced after the paroxysms, and moderates as the latter are farther apart. Thus epileptic dements may be observed presenting intervals of relative lucidity. It is even possible that a patient who for years has been unable to have intercourse with the external world may recover at least a portion of his intellectual faculties when the attacks are suspended or occur at longer intervals. Whitcomb cites the case of a woman who regained her reason after nineteen years, the attacks having ceased subsequent to her recovery from rheumatism. But this is not always the case; in some instances the dementia progresses steadily, whether the par-

oxysms cease or not. It would be proper, therefore, to distinguish two forms of dementia—one due to the exhaustion consecutive to the seizures and subject to remissions, and another definitive form without ameliorations which may be refered to the cerebral lesions observed in epileptics and which should be called degenerative dementia.

Besides the late intellectual degenerations in epilepsy, other mental troubles are met with, such as idiocy, which are connected with an arrest of development of the nervous system.

It is admitted that one-third of all idiots are epileptics, and yet this proportion does not include the convulsions of infancy which are so frequent in idiots. The two diseases may undoubtedly depend upon the same cerebral lesions, but idiocy is often aggravated by epilepsy, and it is only when we succeed in suppressing or abating the frequency of the attacks that we can exert a favorable influence on the development of the intelligence. Moreover, has it not been observed that education offers the best chances of success in epileptic idiots?

It should be added that in a fair number of epileptics the mental state presents nothing abnormal and there are some even who exhibit no stigmata of degeneration. Great men are known to have been affected with this disease, and it is not rare to meet in society persons in whom epilepsy manifests itself solely by convulsive attacks without any influence upon the psychical condition.

Epilepsy doubtless is to be observed in perfectly developed persons whose nutrition is excellent and who are free from important malformations. But it is not safe to accept as true the opinion of Lombroso, who claims that "among epileptics we encounter the great height, the greater weight than the average, and the well-nourished condition so often noticed in moral lunatics and in born criminals."

The anomalies are so much the more numerous and important as the disease has manifested itself at an earlier age and under the influence of less active exciting causes. The importance of the morphological or functional anomalies is therefore in direct relation to the intensity of the predisposition; from the point of view of the diagnosis and prognosis of epilepsy their study is not without interest, but we must take care not to mistake for signs of degeneration peculiarities which may constitute racial characteristics but not teratological malformations.

The ancient authors already noted that deformities or physical defects were frequent among epileptics. Dumas de Montpellier described the inferiority of the facial angle; Solbrig, the narrowness and deformity of the vertebral canal. Lasègue studied with care the

cranial asymmetry which he considered as constant in true epileptics. According to him, the malady is the result of this malformation, which is effected at the period of osseous consolidation. This view includes much exaggeration. Craniofacial asymmetry is very frequent in epileptics, but its absence does not preclude the diagnosis of epilepsy, and moreover it is not characteristic of this disease, for it is often observed in healthy persons. As to whether epilepsy is the consequence of this malformation or whether both result from the same disturbance of evolution is an open question, and we must content ourselves with the statement that this asymmetry and epilepsy frequently coexist. Besides, Guéniot and Parrot have shown that it may be artificial and due to the lateral propulsion of the cranium from lateral decubitus seen in all young children.

Usually the asymmetry affects the cranium and the face at the same time. The frontal eminence is less marked on one side, the parietal eminence is smaller, the orbit is less wide and not so high, and the malar bone is less prominent. The nose deviates, the facial muscles are less developed on the same side, and the lines of the face, in the absence of all paralysis, are less marked. In very pronounced cases the body of the inferior maxilla is shorter and the teeth are often badly placed on the atrophied side. The vault of the palate may be highly arched.

Among the cranial deformities we also often observe either a relative increase of one of the diameters, for instance, the vertical (dolichocephaly, sugar-loaf head), or an absolute increase or diminution of all the diameters (macrocephaly, microcephaly).

It will be sufficient to give a simple enumeration of some of the anomalies which have been observed and which I have reviewed elsewhere:" On the part of the skeleton—asymmetries of the thorax and of the pelvis, funnel-shaped chest, unequal length of the extremities, relatively abnormal length of the fingers, syndactylism, etc.; on the part of the sense organs—different color of the two irides, asymmetry of pupils, corectopia, the auricle is often deformed in its entirety or in its different parts; anomalies of the genital organs are especially frequent, as are also those of the skin."

The nutrition of these patients is often defective. Beau noted that epileptic girls menstruated late. They frequently present disturbances of the general health without apparent cause, such as crises of weariness, digestive troubles, and diarrhœa with a saburral state. Metabolism is less active than in healthy subjects; the proportion of hæmoglobin in the blood is frequently below the average even in the intervals of the paroxysms. Respiration is often insufficient, and it is not rare to find the vital capacity of the lungs diminished in the

absence of pulmonary lesions. The organic deterioration of epileptics has been further elucidated by the researches of Moreau de Tours, who has shown that epilepsy often coexists with chlorosis, tuberculosis, and scrofula. The state of depression which in these patients augments after each paroxysm is more or less present in the intervals and manifests itself by a longer time of reaction and a slighter muscular energy than in normal persons. Several movements which may be looked upon as late acquisitions, like the isolated flexion of the last phalanx of the thumb and the rotation of the forearm, are often defective.

The disturbances of sensibility are much less rare in epileptics than was formerly believed. Sensibility is often blunted; of this we may convince ourselves, with Batigne and Ouvry, by studying the pressure sense;[19] analgesia is frequent. Acuity of vision is often diminished; in one-third or one-fourth of the patients we observe a concentric limitation of the visual field. Dyschromatopsia is not rare and complete achromatopsia has been noted.

Gustation and olfaction are likewise reduced, as I have shown in connection with Batigne and Ouvry,[20] and the same is true of audition.

We need not expect to find all these vices of development in every epileptic, but they are important in some degree in harmony with the relative predisposition. They are most pronounced in hereditary epilepsy. The search for all these stigmata, therefore, is one which should be made in detail with every patient if the diagnosis and prognosis are to rest on a sure foundation.

Etiology.

The exciting causes of epilepsy are very numerous, but they act merely upon a prepared territory, whether it be through an irritative lesion in the partial variety, or through a state of irritable weakness of the nervous elements of impaired development in epilepsy without apparent lesion.

This morbid irritability is met with in a large number of the members of the teratological family. It is transmitted by heredity, but rarely in the same form; dissimilar or transformed heredity is much more frequent than similar heredity.

Dissimilar heredity has long been recognized in epileptics, and the authorities who have treated of the subject mention among the antecedents of epilepsy alienation, hysteria, suicide, somnambulism, chorea, general paralysis, etc. Drunkenness is to be considered as an important factor (Moreau de Tours, Hammond, Nothnagel, and others). One proof of the relationship of these maladies with epi-

lepsy is furnished among others by their frequent coexistence in the same person. The study of the progeny of epileptics is no less interesting in this respect. Among their offspring are many vesanics, idiots, etc.

Similar heredity in epilepsy, on the other hand, has been considered to be extremely rare by numerous authors (Beau, Morel, Delasiauve, Lasègue, and others). Louis has even denied it. Still among the older writers (Boerhaave, Van Swieten) epilepsy is often found mentioned in the ascendants of epileptics. It has been again described by recent writers (Moreau de Tours, Voisin, Echeverria, and others). We cannot say, therefore, that similar heredity in epilepsy is rare; it must simply be considered as less frequent than dissimilar heredity.

Hereditary transmission of epilepsy may be direct or indirect, that is to say, it may be effected through the ascendants or the collaterals. According to the statistics collected by Echeverria, Bourneville, and myself, similar heredity seems to be more frequently indirect. Atavistic heredity is often observed, the malady passing from the grandfather to the grandson without affecting the son. Sometimes it happens that epilepsy manifests itself at the same age in two successive generations (homochronous heredity); but in general anticipation occurs in the descendants, that is to say, they are attacked at an earlier age than the ascendants. Under such circumstances it may even happen that the disease manifests itself in the son before its appearance in the father.

An important part has been ascribed to consanguinity as a congenital predisposing factor to epilepsy (Trousseau, Boudin); morbid consanguinity may give rise to all sorts of degenerations and to epilepsy in particular, but there is nothing to prove that the same result would follow the marriage of kindred parents free from every pathological taint.

Among the causes affecting the ascendants may also be mentioned a disproportion in the age of the parents, especially a greater age of the mother. Too early or too late unions have also been given as causes.

Besides family taints in the ascendants, there are acquired conditions which may be considered as important factors in the epilepsy of children; for instance, excesses, acquired syphilis, poisoning by lead or mercury, etc. Still we should not consider as independent of every hereditary taint such voluntary intoxications as alcoholism and morphinomania. The majority of patients in this category are neuropathic and impulsive.

In certain cases it has been possible to trace back the epilepsy of

children to absolutely transitory states in one or both of the parents, whether at the moment of conception or during gestation. Numerous authors have dwelt on the influence of drunkenness at the moment of conception (Esquirol, Morel, Lucas, and others). Bad hygiene, defective alimentation, infectious diseases of the father and mother at that moment are also predisposing factors.

All these debilitating causes may act in the same way through the mother during pregnancy. Aside from every anterior neuropathic taint, emotions of the mother during that condition have been particularly inculpated. It is pertinent to ask if the neuropathic state of the mother is not to be taken into account in cases cited in corroboration of this opinion, for in nearly every instance there were terrors produced by slight causes out of all proportion to the intensity of the emotions. Be that as it may, it cannot be denied that the fœtus reacts strongly under the influence of emotions of the mother, and must necessarily participate in the convulsive movements of the latter. Investigations by Dareste and myself have shown by what slight influences the development of organs may be inhibited, especially during the earliest period of embryonal life. This fact may be taken in connection with the frequent existence of mental disturbances in subjects of illegitimate birth.

At the time of delivery and during the first weeks of life certain more or less problematical causes have been advanced as capable of producing epilepsy in the infant; for example, the application of the forceps, long retention of the head in the pelvic cavity, and asphyxia due to constriction of the funis. Certain hygienic faults have likewise been incriminated, such as the habit of placing the infant always on the same side, as likely to produce an obliquely oval deformity of the cranium, and the use of the close-fitting caps, in countries where they are still employed.

While the child is at the breast, defective hygiene, emotions, and especially alcoholism in the nurse have been said to be liable to produce a predisposition to epilepsy. I merely enumerate these causes without dwelling upon them, since their influence is often difficult to demonstrate positively.

Can acquired epilepsy of the parents be transmitted to the descendants? Certain experimental facts seem to prove it. Thus Brown-Séquard has shown that guinea-pigs, which have been artificially rendered epileptic, may give birth to young affected with convulsions. I myself have observed a man who had become epileptic by traumatism and who seemed to be free from hereditary taint. He recovered later; but while he was subject to convulsive attacks he had a daughter who became epileptic at the age of five years. Ad-

ditional closely observed clinical facts are needed to show that no earlier predisposition was present in the ascendants.

Nearly all authors admit that epilepsy is more frequent in women, but the statistics upon which they base this opinion are not available for comparison.

No age is exempt from epilepsy; it may occur from the earliest infancy, during which convulsions are so frequent, to advanced age in which the disease is much less rare than is generally believed. Lasègue claimed that true epilepsy appeared only between the fourteenth and twenty-eighth years of life. It has been asserted that after twenty years hereditary epilepsy is no longer to be feared (Nothnagel, Echeverria).

This latter opinion is erroneous, for we know now that epilepsy may supervene at an advanced age, and in these cases we frequently meet with an hereditary taint, a predisposition which could manifest itself only under the influence of bad hygiene, excesses, or emotions. Late epilepsy is more frequent in women, and the menopause seems to play an important part in its appearance.

The distinction between idiopathic and symptomatic epilepsy is arbitrary; clinically it is often impossible. Not less arbitrary is the effort to make a separate category of eclampsia or to differentiate the convulsions of early infancy from those which develop in the course of scarlatina or in the puerperium.

Besides, there is no longer any hesitancy nowadays to connect the convulsions of infancy with epilepsy. The identity of the two affections is superabundantly proven, not only by the similarity of the symptomatic pictures, but also by the hereditary influence and by the frequency of infantile convulsions in the early history of confirmed epileptics. Baumès says: "Between the eclampsia of infants and epilepsy there is only a difference in the course which time alone can establish." Why then distinguish infantile convulsions from epilepsy and say that epilepsy is rare or does not exist in children? In them, on the contrary, it is most frequent. I have been surprised to find that Walton and Carter assert that when children who have had convulsions are cured and have remained so for some time, they possess no greater liability to epilepsy than others.

There is still less unanimity as regards the eclampsia of adolescents and adults. In renal affections complicated with uræmia the convulsions are attributed to a poison circulating in the blood and exerting an irritant action upon the central nervous system (uræmia, ammoniæmia, creatinæmia, etc.); but it may be asked if this is not simply an exciting cause acting upon a neuropathic subject who is predisposed to cerebrospinal reactions. In fact, the varied clinical

forms of uræmia would lead us to suppose that since all persons do not react alike to the same blood alteration they must present different organic predispositions. I am convinced that the eclampsia of infectious diseases and that of the puerperium are morbid manifestations of the same order as ordinary epilepsy.

If we inquire closely into the antecedents of patients who are attacked by eclampsia, whether during convalescence from scarlatina or during pregnancy or at the moment of delivery, we can in many cases discover a neuropathic hereditary taint, or we may learn that they have previously suffered from convulsive seizures of an eclamptic form during dentition, gastrointestinal disorders, infectious diseases, etc. It is safe to say that a similar investigation has been especially neglected by observers. But, on the other hand, eclampsia does not always constitute a temporary accident; it may be replaced subsequently by ordinary epileptic fits. It would then be correct to say that the acute epilepsy has passed into a chronic condition. The morbid state existed previously and needed merely an occasional cause to be called forth; indeed, Gowers had already made the remark that scarlatina is the disease which is most frequently followed by epilepsy. I have observed epilepsy become definitely established after puerperal eclamptic seizures. Finally, the study of the descendants of eclamptics is no less interesting in this respect, showing that the neuropathic condition may be transmitted to the children in variable forms.

There are, therefore, acute forms of epilepsy, eclamptic epilepsies, which are brought about by certain pathological or physiological conditions; but, like ordinary epilepsy, they develop only in consequence of a neuropathic predisposition evidenced by anterior hereditary or personal accidents. These acute epilepsies of infancy, of the puerperium, etc., may terminate in recovery, leaving the organism with a tendency to occasional convulsions, or they may pass into the chronic state and be transformed into ordinary epilepsy.

The disease is observed in all countries, but the reported statistics are not sufficient to enable us to judge of its relative frequency. The part played by climatic and seasonal influences is entirely unknown.

Among the physiological conditions which may act as predisposing causes the genital function should be mentioned in the first place. In women the influence of puberty has been referred to many times; it is especially manifest when menstruation is established with difficulty. Moreover, the menstrual functions in epileptic women often coincide with an exacerbation of the fits, and the disease then progresses in a markedly periodical form (Gowers, Voisin). Epilepsy

may also appear or become aggravated by the onset of dysmenorrhœa or suppression of the menses. In one of Somers' cases epilepsy was connected with an obstacle to the evacuation of the menstrual blood due to imperforate hymen, and ceased after incision. The menopause has often been inculpated, and I have described above, in connection with tardy epilepsy in women, the appearance of convulsive attacks at this time of life. It is quite certain that these causes become effective only in predisposed subjects, and this remark applies more or less strictly to all the exciting causes.

The influence of pregnancy, from this point of view, is extremely variable. Sometimes it induces the appearance of attacks which do not recur after delivery. At other times it aggravates the pre-existing disease, but the exacerbation is merely temporary. In a certain number of cases, however, the aggravation of the disease persists after the puerperium. On the other hand, cases have been reported in which the influence of pregnancy was favorable. Thus the fits have been observed to become milder and less frequent during gestation and this improvement has persisted indefinitely. In a few cases, unfortunately but too rare, pregnancy seemed to suppress the attacks which thereafter never recurred. Sometimes we may observe during pregnancy other epileptic disturbances than convulsions; thus, Evans described facial neuralgia coinciding with convulsive seizures.

I need not revert to labor and delivery as exciting causes of the fits after what I have said as to the nature of eclamptic convulsions, so called. But, in passing, I may point out how erroneous it would be to apply the name eclampsia to all the convulsions which are manifested at the time of parturition. Frequently hysteria is likewise called forth at that moment, and in this way we may account for the benignancy of certain morbid states which are called eclamptic and are even accompanied by elevation of temperature when the convulsive crises have been numerous.

A certain number of chronic intoxications are capable of provoking epilepsy. Alcoholism should be cited first in the list. Chronic alcoholism acts undoubtedly by way of causing debility, and it may play a prominent part in heredity. But it is claimed as a fact that acute alcoholism is a powerful exciting factor of an epileptic fit. It has not been proved that it may act upon a person free from every predisposition; but it is a matter of daily observation in asylums that exacerbations of the attacks occur in epileptics after they have been permitted to go out. Some of these patients are so extremely susceptible that one or two glasses of wine suffice to bring back the paroxysms; rarely need they go as far as complete drunkenness.

Maisonneuve noted that drunkenness could reawaken an epilepsy which had been apparently cured for years. The fit may occur during the period of alcoholic excitement or it may be delayed until the stage of depression on the day following the excess.

Absinthism acts in the same way, in all probability. The proof has never been furnished that absinth is capable of exciting epileptic attacks in a person who is free from every nervous taint. The convulsions which have been caused in the lower animals by absinth intoxication do not constitute a sufficient argument. Besides, the influence of this poison is very difficult to determine with precision in man, since those addicted to it absorb nearly always a certain amount of alcohol with it. According to Cadéac and Meunier, of Lyons, the evil effects produced by this liquor are not due alike to all the essences which enter into its composition. The essence of anise in particular is of the greatest toxicity, while the essence of absinth is said to be almost harmless.

Plumbism forms a favorable field for the development of epileptic manifestations (Tanquerel des Planches, Leuret, Grisolle, and others). Intoxication by lead is a powerful exciting factor of nervous disturbances of every variety. It often produces acute epilepsy in serial attacks with delirium, that is to say, the status epilepticus, which may terminate in death. Aside from this, plumbism may give rise to an hereditary predisposition by the sclerous brain lesions which it causes.

Among the toxic agents which have been cited as able to evoke epilepsy may also be mentioned chloroform, ether, morphine, opium, tobacco, cocaine, ergot, etc.

In predisposed subjects the infectious diseases may determine the appearance of epilepsy. Reference has been made above to scarlatina; it is mainly by the frequent renal complications that this malady causes epilepsy. Uræmia, whatever its origin, often gives rise to convulsions, and I have stated above the part which should be assigned to the substratum, *i.e.*, to the morbid predisposition, in manifestations of this class. The other eruptive fevers, especially typhoid, belong in the same group.

The influence of paludism was noted by the older writers. It has been claimed that the epileptic fit may replace the fever or alternate with it. On the other hand, Selade held that paludism might have a favorable influence upon epilepsy.

Syphilis is bound to keep an important place among the chronic affections which are capable of causing epilepsy. In the secondary stage it may give rise to convulsive attacks independent of any known anatomical lesion. Sudden general attacks (d'emblée) may super-

vene during this stage and may be observed to disappear under the influence of specific treatment. In other cases pre-existing epilepsy has been aggravated by syphilitic infection, and mercurial treatment has got the better of this exacerbation. However, it may be that the aggravation of epilepsy persists in spite of the specific medication, and that bromide of potassium alone has any marked influence upon the attacks.

A person affected with syphilis may have a predisposition to epilepsy which manifests itself only at an advanced age, entirely independent of the syphilitic infection. Losing sight of the possibility of the tardy appearance of epilepsy Fournier has endeavored to establish a parasyphilitic epilepsy to which, however, he has been unable to assign a single special characteristic.

But it is especially during the tertiary stage of syphilis that epilepsy shows itself with particular frequency. It may then be dependent upon the most varied lesions, such as gumma of the brain, lesions of the cranial bones, of the meninges, of the cerebral vessels, etc. Syphilis is one of the oldest-established causes of epilepsy, but its cerebral lesions have been studied from an anatomical and clinical point of view especially in recent years (Buzzard, Hughlings Jackson, Charcot, Fournier, and others). Does the existence of material lesions here exclude the influence of all neuropathic predisposition? It seems not; for a large number of syphilitics suffering from epilepsy belong to neuropathic families. There can be little doubt that overwork, physical or mental, and the struggle for existence contribute to the cerebral localization of the lesions.

The morbid predisposition may remain in a latent state for a longer or shorter time, when it suddenly manifests itself after an apparently insignificant cause, such as indigestion or an affection of the ear. It seems that there is in cases of this kind an epileptogenic zone which varies in each individual, but whose irritation is usually sufficient to bring on the attack.

Thus epilepsy has sometimes followed wounds of nerves. It was by irritating or cutting the sciatic nerve that Brown-Séquard succeeded in rendering guinea-pigs epileptic. Certain painful irritations such as neuromata seem to have been the cause of convulsive attacks in man. In confirmation of this view is the fact that the attacks ceased after the extirpation of the cicatrix or the tumor. When traumatic lesions of a nerve are present, sometimes a very slight irritation of the skin, either in the distribution of the nerve or elsewhere, suffices to bring on an attack. For instance, in a patient suffering from left sciatica resulting from a severe contusion of the thigh, pinching the skin of the neck on the left side was suffi-

cient to produce an epileptic fit. Such cutaneous epileptogenic zones may be present in trigeminal neuralgia at the level of the painful spots. Furthermore, the existence of epileptogenic zones on the surface of the body, aside from any local or distant nerve lesion, has been described. Mere contact of such zones may suffice to provoke a fit. They are present chiefly on the head, the temple, the ala nasi, the lip, about the internal canthus of the eye, and also on the skin of the neck and on the hand.

Epilepsy may appear in connection with dental disturbances. The frequency of convulsions in young infants during dentition is well known. In adults we sometimes observe the coincidence of the attacks with the eruption of a wisdom tooth or with a toothache. Epilepsies of a reflex nasal origin have been described, which were caused by the presence of worms in the nares (Sauvage), or by foreign bodies in the frontal sinuses (Legrand du Saulle). In infants nocturnal attacks may be due to impaired nasal respiration (Kjelman).

Irritations of the auditory nerve are particularly prone to provoke epileptic fits. They may be caused by the presence of foreign bodies or insects in the external auditory meatus. Magnin and Nocart have shown that epilepsy in dogs is often due to the presence of ascarides in this canal. Here likewise cure often follows the extraction of the foreign bodies. Still the disease may persist when consecutive lesions of the ear have developed. Auricular epilepsy is often met with in consequence of ordinary inflammatory lesions of the auditory canal and the Eustachian tube. Chronic otitis may be complicated with convulsive attacks, and these may disappear with the lesion. All forms of paroxysms are observed in consequence of lesions of the ear; vertiginous attacks, however, are most frequent. Indeed, if the predisposition in patients suffering from auricular vertigo were inquired after it would often be found. Besides, Peugniez and Fournier have shown that mental stigmata of degeneration are frequent in persons affected with auricular vertigo. Goodhart also admits that the disease develops on a neuropathic basis.

Errors of refraction, necessitating as they do efforts of accommodation, may cause epilepsy.

Epilepsy has been ascribed, too, to lesions of the respiratory apparatus, such as laryngitis, traumatism of the larynx (Borie), and pulmonary affections, especially those of the pleura. Operations on the pleura, such as injections and particularly pleurotomy, are liable to provoke convulsive attacks.

It has long been known that irritations of the abdominal sympathetic may cause reflex convulsions. A great many authors have in-

cluded affections of the stomach among the causes of epilepsy. Simple indigestion even has been inculpated and in such cases good results have been obtained by the use of emetics (Delasiauve). In young infants enteritis and diarrhœa frequently occasion the appearance of convulsions. The presence of foreign bodies, especially worms, in the intestine has been repeatedly described as a cause. Trousseau cites several cases of epilepsy due to tapeworm. Lumbricoid worms and oxyurides may give rise to the same accidents. The convulsive crises may cease after the expulsion of the parasites, but not rarely they persist.

Mention may also be made of hepatic colic and affections of the genital apparatus—the testicle in man and the uterus in woman—as occasional causes of epilepsy, and the influence of menstruation on the periodical course of the seizures should be borne in mind.

General or partial epilepsy constitutes one of the fundamental symptoms of cerebral tumors—syphilitic gummata, tubercles, various neoplasms, exostoses, abscesses, etc. The predisposition, which can be constantly traced in addition to the predominating causes enumerated thus far, seems here less essential; still it is certain that lesions of the same nature and similarly located are accompanied by convulsions in one case and not in another. Sometimes the lesion itself seems merely to create a tendency to the disease, and the epilepsy supervenes only in connection with some moral or physical shock. An instance of the kind is reported by Gowers: A child in perfect health swallowed a slate pencil; some hours afterward it had a severe epileptic fit; two months later it died and a glioma was found in the pons.

Traumatic lesions of the cranium and the cerebrum may produce epilepsy independent of every predisposition. Sometimes the disease follows a violent shock, some concussion not accompanied by either wound or fracture. Westphal succeeded in rendering guinea-pigs epileptic by striking them repeated blows on the head. The influence of traumatism is clearer when there is a fracture with depression of the fragments and cerebral compression. In such cases the onset of the convulsions may be immediate and they cease after the fragments are lifted. A similar result is sometimes produced by an intracranial effusion of blood; evacuation of the fluid may relieve the attacks immediately.

Lesions of the spinal cord also, though more rarely, may cause epilepsy. It may be recalled that Brown-Séquard observed the occurrence of epilepsy after various experimental lesions of the cord, such as complete transverse section of the posterior columns and of the posterior horns, section of the lateral or anterior columns, and

simple puncture. The guinea-pigs experimented upon presented spontaneous epileptic fits, and attacks were also produced by irritating certain cutaneous zones of the face and neck. In man a number of cases have been observed in which epilepsy was due to compression of the cord by traumatism, by a tumor, and by some vertebral affection. Chipault cites the case of an infant suffering from Pott's disease, which began to have epileptiform attacks from the moment that a fistula allowed the escape of the cerebrospinal fluid.

This refers to ordinary epilepsy, produced by spinal lesions. But Brown-Séquard has described a different affection under the name of spinal epilepsy, which manifests itself when the continuity of the cord is broken in one of its halves. It consists of clonic and tonic convulsions which are sometimes very violent and may occur spontaneously, but are more frequently excited by cold, friction of the muscles, and cutaneous irritations.

The affection should be brought into the same category with epileptoid tremor, the occurrence of which in man has been described by Charcot and Vulpian and is observed in a large number of diseases, namely, slow compression of the cord, amyotrophic lateral sclerosis, hypertrophic cervical pachymeningitis, and hemiplegia with contracture; in a word, in all cases characterized by a secondary or primary degeneration of the lateral columns.

The efficiency of the causes which have been given as having produced an attack of epilepsy is very often problematical. But the laity has at all times ascribed great importance to emotions, especially fright, to falls, blows, etc. It is natural that the patient or his family should seek to explain the attack by an antecedent occurrence, rather than to admit an hereditary taint. Frequently enough, however, we can ascertain by minute inquiry that the fact in question is without importance or that it has taken place in the remote past.

It is, however, beyond doubt that in predisposed subjects a fit may be immediately provoked by a more or less intense shock of the nervous system. Emotions have been spoken of above: in particular the asthenic emotions are given as frequent occasional causes (grief, fear, and prolonged anxiety). These psychical states, moreover, are associated with an overexcitability which is very favorable to the evolution of convulsive phenomena. At the menstrual periods these emotions act even more powerfully by reason of the nervous susceptibility which accompanies this physiological condition. The convulsive discharge sometimes follows immediately after the emotion has been experienced; but this is not always the case. The nervous depression

succeeding moral shocks may last some time and the attack may supervene at a given moment during this asthenic state, occasionally rather long after the initial cause.

An epilepsy which has manifested itself in connection with some fright may become definitely established and the fits may thereafter appear without exciting cause. In other instances the attack recurs invariably when the same exciting influence becomes active; for instance, the sight of a corpse, of a precipice, of blood, etc. Sometimes, however, the fright is merely the prelude of the attack, and it is only the result of a terrifying visual hallucination which is a part of the aura. For instance, the patient thinks he sees a wagon rapidly driven towards him; the fear of being run over makes him fall unconscious; but nothing in reality corresponds to this vision. Here the emotion is really a part of the paroxysm, but the patient remembers it and attributes the attack to it. In the same way terrifying dreams have been incriminated. It is reasonable to suppose that the hallucinations during sleep bear some relation to the frequency of nocturnal attacks in certain patients.

Sexual excitement has seemed in some cases to have been productive of a seizure. Some epileptics have been known to fall into a fit at the moment of coition or soon thereafter.

Certain patients are attacked while making a violent effort; others during digestion, even when it proceeds normally. In this connection we may recall the congestive epilepsy of excessive eaters which Lépine mentions.

As to the causes which influence the periodical return of the fits or their frequency, aside from menstruation in women, nothing definite is known. The influence of the lunar cycles, of the direction of the wind, of climate and seasons is very problematical. A matter of greater interest is the study of the variation in the number of the fits during the waking and sleeping hours. I was able to demonstrate at Bicêtre not only a greater frequency of nocturnal attacks, as above noted, but also a relative predominance about nine o'clock in the evening or about three, four, or five o'clock in the morning. The morning recrudescence of the seizures is possibly the consequence of an accumulation in the blood during the night of convulsive substances (Bouchard). Or possibly it is the cold of the early morning hours which plays a part.

All the causes already described as capable of producing epilepsy may also influence the return and the frequency of the paroxysms. It is a fact, however, that the patient is liable to have, at least apparently, spontaneous exacerbations and ameliorations.

As to the influence of intercurrent affections I can only repeat that

it is very variable and does not enable us to deduce any therapeutic indications or to advise the injection of microbic toxins."

Diagnosis.

Epilepsy may manifest itself by so manifold and varied symptoms, as stated above, that the diagnosis is often difficult and requires a minute clinical examination as well as a thorough familiarity with all the forms of the paroxysms. When it presents itself in the form of major convulsive attacks having the characteristics described above, we may very often express a categorical opinion, especially after having witnessed a paroxysm. But this is by no means always the case; as a rule we have to be satisfied with an interrogation and inspection of the patient. The stigmata of predisposition, hereditary neuropathic taints, anatomical and functional signs of degeneration, will barely serve as a basis for a suspicion unless there are certain cranial malformations.

The epileptic may be entirely ignorant of his disease when the convulsive fits occur exclusively during the night, but ordinarily he experiences on awaking a heaviness of the head with a more or less marked confusion of the intellect or a hesitancy of speech. Besides he is apt to have had during the night an involuntary evacuation of urine or fæces. It is found that the tongue is swollen and has been bitten; sometimes there is on the face or neck a small point of hemorrhagic extravasation. Occasionally the patient even presents traces of bruises, the cause of which he does not know.

The description of eye-witnesses of the fit, provided they have no interest in practising deceit, may at times suffice as the basis of an opinion; but this is only when they clearly and spontaneously state the principal major phenomena, *i.e.*, pallor of the face, sudden fall, convulsions, traumatism, involuntary evacuations, stupor, etc. When the physician examines the patient a short time after the attack, the determination of the phenomena of depression is of great value. But the convulsive crises are not so constant nor so characteristic in themselves that even their direct observation by an experienced clinician can always clear away every doubt.

The major convulsive epileptic fit and the major attack of hysteria present a certain number of features in common. Either one may be preceded by remote precursory phenomena which are not without analogies, *i.e.*, sadness, irritability, restlessness, abrupt movements, anxiety, heaviness of the head, etc. It has been said that previous to the attack hysterics are more expansive, epileptics more gloomy; but there are no precise reliable limits. Male hysterics in particular

often present melancholic manifestations. The immediate indications of the attack also resemble each other in some cases; thus the sensation of a ball rising from the epigastrium to the throat, which is accepted as a characteristic of hysteria, is far from rare in epileptics, even those of the male sex. Still when the sensation starts first from the ovary, and when this organ is at the same time especially sensitive, it appertains almost exclusively to hysteria. Testicular sensibility, on the other hand, is not very rare in male epileptics. A sense of oppression or precordial anxiety may inaugurate either attack. A certain number of visual disturbances, such as dazzling, photophobia, and erythropsia, are met with in the prodromal stage of both diseases. The other peripheral auras belong rather to epilepsy.

The major hysterical attack begins with an epileptoid stage which is rarely accompanied by an initial cry, and pallor of the face is exceptional in it. But the convulsions faithfully reproduce the tonic stage of the epileptic fit—lateral torsion of the head, twitching of the eyelids, rapid grimaces, tremulous rigidity of the extended limbs. This tonic stage is followed by the great movements of the so-called clownish stage in which the arching of the body and jerky protrusion of the belly occupy a prominent part, then come the passionate attitudes with delirium.

The distinction is easy when the phases of the major hysterical attack follow in their schematic order, but often it is otherwise. The effect of ovarian compression may then furnish valuable assistance; it has no influence upon the epileptic fit, but in hysteria it often cuts short the attack or at least modifies its intensity considerably. Not rarely, however, this compression as well as that of the testicle in man remains without effect in hysteria. We must also take into consideration the characteristics of the epileptiform attack; the great movements, the propulsive movements of the abdomen, and the twitching of the eyelids belong chiefly to hysteria. Account should also be taken of the absence of initial pallor of the face, of the longer duration of the hysterical fit, of the less marked consecutive depression, of the tears and bursts of laughter which often occur at the end of the attack. Lastly, the presence of permanent stigmata will be subsequently searched for, *e.g.*, sensori-sensorial hemianæsthesia, ovarian tenderness, painful spots, etc.

The diagnosis of the nature of the crisis, however, may present extreme difficulty in certain patients who are distinguished by the name of hystero-epileptics. It is admitted that the two neuroses may coexist in the same person, develop side by side without influencing each other, and give rise to distinct crises (hystero-epilepsy with separate crises). There is nothing improbable about this, for

we know that hysteria may be associated with all kinds of neuropathies, but we are justified in saying that the criterion is often lacking by which we could distinguish the epileptic crises in attacks which may be simply incomplete major seizures of hysteria. Lasègue has remarked that in a large number of hystero-epileptics the crises are sometimes more of an epileptic, sometimes more of an hysterical nature.

Cathelineau and Gilles de la Tourette thought they had found in the urine a chemical test, but their hopes have not been realized. Bromide of potassium, which often but not invariably is very effective in epilepsy and seems powerless in hysterical crises, can be considered at best as but an unreliable differentiator.

Like epilepsy, hysteria may manifest itself in the form of serial attacks, the status hystericus, the diagnosis of which is sometimes very difficult. The subintrant attacks may consist solely of the epileptoid phase, and contrary to what has been stated, this variety of the disease cannot be recognized by the form of the convulsions. The difficulty becomes still greater when the temperature rises; thus far this pyrexia had always been considered as belonging properly to epileptic fits, but recent facts have shown that the temperature may reach from 40° to 41° C. (104° to 106° F.) in the course of frequently recurring hysterical attacks and may remain at this point for several days.

The paroxysms of hysteria minor are more easily distinguished from epileptic fits of the same class; they consist of more or less arrhythmical and irregular spasms or incoherent gesticulations not accompanied by pallor of the face, nor by a bitten tongue, nor by complete loss of consciousness, nor by consecutive stupor. Instead of an initial cry they are associated with noisy vociferations; they are often followed by fits of laughing or crying, by abundant micturition, and in all cases by a rapid return to the normal condition without exhaustion. The cephalic congestion which accompanies the hysterical attack rarely imparts to the skin that livid hue which is so frequent in epilepsy.

The two disorders, however, may not resemble each other solely by their convulsive manifestations. Vertigo may be observed in hysteria as well as in epilepsy. This is true also of the ambulatory automatism. Apoplectiform attacks are common to both neuroses.

The isolated or localized spasms which are observed in incomplete seizures or in attacks of partial epilepsy may be mistaken for spasmodic movements of a different nature, such as the tics or choreiform movements, and the reverse. But the convulsive movements of the tics are not accompanied by any kind of clouding of the intellect nor

by pallor of the face, and they leave no stupor behind them. As to chorea, the evolution of the disease and the absence of the phenomena described above suffice for the differentiation.

When epilepsy presents itself in its incomplete, attenuated, or abnormal forms, it is apt to be mistaken for numerous affections. The symptom complex of true apoplexy and that of apoplectiform epileptic attacks may be identical, and the distinction would be impossible if the conditions under which the attack is produced were not taken into account, if the antecedents of the subject were unknown, if the subsequent symptoms were not observed, etc.

The vertiginous forms are not always readily distinguished from syncope; there are the same fall and the same immobility with suspension of voluntary movements. However, in syncope the pulse is imperceptible, while it is strong and vibrating in the epileptic fit. In epilepsy, too, we observe vertiginous accidents with a sensation of rotation or propulsion. Yet this form of vertigo is met with also in disseminated sclerosis, in locomotor ataxia, in lesions of the cerebellum, etc. We must try to ascertain, therefore, if it is not symptomatic of one of these maladies. Finally, before deciding on the epileptic nature of the affection we must have eliminated all the other forms of vertigo, such as those associated with anæmia, neurasthenia, etc. One fact which often complicates the diagnostic difficulties is that epilepsy may coincide with various neuropathic or psychopathic disturbances, such as exophthalmic goitre (Ballet, Oliver, and others), chorea (Althaus, Hawkins, and others), locomotor ataxia (Bernhardt), disseminated sclerosis, etc.

The greatest difficulties are encountered in the diagnosis of the psychical manifestations of epilepsy. When a convulsive attack occurs during one of the flights from home or during an impulsive delirium, there is no room left for doubt; but when the delirious act takes place apart from every manifestation of the kind, its nature is much more difficult to clear up. The states described as emotional inebriety are characterized, like all the psychical paroxysms of epilepsy, by the abruptness of their onset, the consecutive symptoms of exhaustion, stupor, prolonged sleep, and more or less complete amnesia; and they occur in general in persons presenting almost the same stigmata of degeneration as the epileptics. Under these circumstances the diagnosis can be positively made only when the same person exhibits motor paroxysms whose epileptic nature is beyond question.

Epileptic mania manifests itself often as a sequel of the fits or after repeated attacks of vertigo. It is characterized by the existence of internal hallucinations which often form the starting-point of the

delirium or of acts of violence. Besides it has been observed that it is less incoherent than the majority of the other maniacal attacks. The patient is able to see and observe everything, and sometimes answers questions correctly; the movements are violent but they are not incoherent or disordered, and when the patient strikes he often does so with remarkable surety.

Finally, epilepsy should not be charged with all the mental disturbances which may be observed in epileptics. They may present alcoholic delirium and other typical forms, such as the delirium of persecution with its characteristic phases. The diagnosis of these combinations is generally very difficult but not impossible. The evolution of the mental disturbances and the influence of the bromide treatment upon the associated epilepsy are valuable guides. In like manner abstinence and sedatives will overcome the alcoholic delirium. Bromide of potassium, which has a marked effect upon the mental disturbances caused by epilepsy, will be inert in a case of systematic delirium developing independently in an epileptic.

Epilepsy having been recognized in a patient, we must determine its cause, and this part of the diagnosis is not always the least difficult one.

A clinical distinction of the greatest utility from this point of view is here forced upon us. Epilepsy may appear suddenly and manifest itself by a large number of consecutive fits and may then not occur again until after a longer or shorter interval of time; or else it shows itself only by a convulsive attack from time to time during many years or during the whole lifetime of a patient. On this account the division into acute and chronic epilepsy has been made.

It may be stated as a general rule that in acute epilepsy the cause is easily determined and often declares itself spontaneously, while it may be very different in chronic epilepsy.

Acute epilepsy or eclampsia manifests itself in the majority of cases in the midst of a train of general or local symptoms which sometimes dominate the scene. It may occur at all periods of life. In infancy it may be the consequence of evanescent, slight disturbances of the general health, or, on the other hand, of serious and irremediable lesions. This shows how much interest attaches to the recognition of the origin of convulsions in infants. They may be due to the pathological evolution of the dental system, to the presence of intestinal worms, or to simple digestive disturbances. No doubt the validity of these causes is questionable aside from the morbid predisposition; nevertheless it behooves us to watch over the mouth and the digestive functions of children suffering from eclampsia. In some cases the convulsions appear in the train of other cere-

bral symptoms which indicate an inflammatory condition of the meninges of the brain. They may then lead to an incurable hemiplegic paralysis or to idiocy, and the epilepsy may persist.

In women child-bearing presents a condition which is favorable to the development of acute epilepsy or eclampsia. It is especially in the last months of gestation or at the time of delivery that puerperal eclampsia manifests itself; but it may appear much earlier, in the first months of pregnancy. The cause should then be traced; as a routine practice, whenever acute epilepsy appears in a woman of child-bearing age the signs of pregnancy should be looked for. In other cases, again, eclampsia does not manifest itself until after delivery. Albuminuria usually accompanies acute puerperal epilepsy, but it is not invariably present and may be lacking quite often. It should always looked be for, not alone during the child-bearing age but in all cases of acute epilepsy, for renal lesions play a prominent rôle in their development.

In this connection it may be incidentally remarked that all serial convulsions which may be observed in the child-bearing age are not necessarily epileptic, as has been largely claimed. States of eclampsia should be distinguished from hysterical or hystero-epileptic seizures. The diagnosis may be very difficult but will be facilitated by the study of the antecedents and by the finding of the stigmata of hysteria.

Aside from pregnancy, acute epilepsy may manifest itself in the adult under the influence of general diseases or intoxications. First in this series scarlatina and plumbism should be cited. Among the renal complications of the acute diseases scarlatinal nephritis undoubtedly causes the invasion of eclampsia most frequently. Albuminuria should be carefully looked for in scarlatina patients and in those recovering from this disease. The diagnosis is sometimes very difficult in masked forms of this eruptive fever.

In plumbism acute epilepsy is associated with the special symptoms of lead poisoning, such as constipation, colic, paralyses, etc., as well as with the cephalic disturbances, drowsiness, hebetude, and delirium. The convulsive manifestations have at first nothing special about them and it is only in the characteristics peculiar to the intoxication, which are easily recognized, that a differential element will be found.

A similar remark applies to the acute epilepsy of chronic nephritis. The œdema, the hypertrophy of the heart with gallop rhythm, and the character of the urine will reveal Bright's disease and uræmia. It should be remarked, however, that when the fits assume the form of the status epilepticus in the course of uræmia, the tempera-

ture may fail to rise and may even sink below the normal; but nowadays rise of temperature can no longer be considered as excluding uræmia.[32]

Acute epilepsy resulting from a cranial traumatism is easily recognized; it occurs most frequently soon after the shock, but its mechanism is not always the same; sometimes it is an osseous fragment, sometimes a sanguineous effusion which compresses the brain. In other cases it results from a meningoencephalitis following the traumatism and is accompanied by fever, delirium, nausea, and vomiting. A spontaneous effusion of blood may produce the same convulsive phenomena; the hemorrhage is then associated with the symptoms peculiar to the apoplectic stroke.

When epilepsy manifests itself by isolated crises at more or less remote intervals, the search for the cause is often much more difficult. If the attack takes place in the course of an acute disease or of an intoxication in a patient who previously did not present any convulsive accidents, the relation of the cause to this effect is evident; it is reasonable to hope then that the epilepsy will be transitory. It is well known that the majority of the eruptive fevers may provoke epilepsy; it has been observed in the course of cholera, typhoid fever, pneumonia, pleurisy, etc. This is true also of acute alcoholism; the recurrence of the crises with each excess indicates clearly the baneful effect of the alcohol. In chronic alcoholism the relation is less evident; but the presence of the ordinary signs of intoxication, trembling, disturbance of speech, insomnia, nightmares, etc., in an epileptic should lead one to think of it, and in a similar case the diminution or cessation of the fits under the influence of abstinence will confirm the diagnosis.

If the patient is affected with syphilis, energetic treatment with mercury and the iodides should be resorted to, for in such cases these drugs furnish a true criterion of the origin of the epilepsy. In fact syphilis, whether inherited or acquired, is one of the infections which most frequently cause epilepsy. Hence even when syphilis does not declare itself by characteristic objective lesions, when the osteocopic pains and the nocturnal headache are absent, it should be suspected whenever epilepsy develops apart from every evident cause, especially in a subject free from marked hereditary predisposition, and the appropriate treatment should be instituted. Fournier has shown, in fact, that syphilis may for a long time manifest itself solely by epileptic fits without any other symptom. The fits which occur under the influence of syphilis have, moreover, nothing special in their types; we may observe vertigo, major attacks, and psychical forms.

It is convenient, however, to consider separately that form of epi-

lepsy which appears in the course of an old syphilis as a result of tertiary lesions of the brain, gummy tumors in particular. In the first place the age of the patient may arouse suspicion. Syphilitic epilepsy appears between twenty-five and forty years, while so-called spontaneous epilepsy generally develops before that period. Moreover, instead of setting in suddenly, in the midst of a state of perfect health, it is preceded by a general modification of the organism, by a state of depression affecting the psychical as well as the other functions. This state consists in a loss of muscular power, a weakening of the memory, an intellectual degradation, and an unfitness for any kind of work. The patient experiences a feeling of profound discouragement on observing his physical and mental powers lessening from day to day. Dyspeptic disturbances are not rare and a notable loss of flesh may ensue. In the mean time the patient experiences now and then some slight hesitation in speech, numbness, and a temporary weakening of the limbs. Some patients are in a continual vertiginous state, others suffer from time to time attacks of true vertigo with loss of consciousness. One of the most characteristic symptoms of this period is a headache generally located in the frontoparietal region, occurring, at least at first, chiefly at night, beginning at a definite point and gradually extending until the whole head is involved, and often giving rise to an insuperable insomnia. Along with this headache may be observed rheumatoid pains in the extremities, sometimes fixed, at other times fleeting. Finally the convulsive attacks appear. Here the disease presents some peculiarities in its course. Thus it rather frequently manifests itself, at least in the beginning, in the form of partial or hemiplegic seizures. The attacks follow each other at shortening intervals and may constitute a true status epilepticus unless treatment be instituted. Above all, however, the etiological diagnosis is based on the knowledge of the patient's antecedents and especially on the results of specific treatment which as a rule rapidly ameliorates the attacks.

The convulsions developing in the course of meningitis do not differ in their form from those of epilepsy. The concomitant symptoms, however, usually enable us to recognize their origin; these are fever, headache, abdominal disturbances, vasomotor phenomena, and the characteristics of the meningitic pulse.

The trembling of the lips and tongue, the intellectual and physical enfeeblement, and the mental disturbances enable us to recognize general paralysis of the insane in which epileptic convulsions are frequent. According to Wigglesworth, atrophy of the papilla is a very early sign, the demonstration of which should have great diagnostic value in the beginning of the disease. Tuberculous meningitis

may likewise give rise to convulsions which sometimes precede the appearance of the most characteristic phenomena, in the child as well as in the adult. It is easy to understand the interest attaching in similar cases to the etiological diagnosis, with reference to the prognosis.

Cerebral neoplasms of every kind, whether tuberculous, gummous, or cancerous, may produce epileptic fits. A general examination of the patient, inspection of the fundus of the eye, a study of the functions of the cranial nerves, etc., will enable us to suspect or determine the nature of the trouble. In two cases of epilepsy associated with multiple lipomata, Letienne and Claus attributed the convulsive seizures to similar tumors in the brain.

When epilepsy cannot be traced to some general or central cause, lesions of the peripheral or visceral nerves may account for it. Traumatic lesions of the peripheral nerves should be searched for with care, and it may be borne in mind that the premonitory aura often starts from the injured point.

In the absence of every other apparent exciting cause the starting-point of the disease may be looked for in some visceral affection, such as intestinal worms, renal or biliary lithiasis, digestive disturbances, etc. The origin of the so-called reflex epilepsies is most frequently obscure and difficult to determine. In women the connection of the crises with the menstrual function may indicate an affection of the uterus or its adnexa. In men, the fits have been ascribed to stricture of the urethra, atresia and excessive length of the prepuce, etc.

Among the nerves of special sense there is one whose lesions should be investigated with great care, and that is the auditory nerve. If we have to deal with a foreign body or a permanent lesion of the ear the diagnosis is easy; but sometimes epilepsy depends on very evanescent disturbances of this organ (Boucheron). One of the most interesting among these is obstruction of the Eustachian tubes with rarefaction of the air in the tympanic cavity and pressure on the labyrinth (otopiesis). The diagnosis is all the more interesting because the treatment is often effective. The crisis of auricular epilepsy is sometimes ushered in by tinnitus aurium and may be accompanied by vomiting, but otherwise there are no special features. More frequently vertigo with loss of consciousness may be observed. The affection described as Ménière's disease or auricular vertigo cannot be categorically distinguished from epilepsy, since in the latter form of vertigo consciousness may be retained, while it may be lost in auricular vertigo. The differentiation of laryngeal from epileptic vertigo is not much clearer; the advocates of the theory of laryngeal vertigo, moreover, admit that it is relieved by the bromides like auricular vertigo."

When the recognized cause of epilepsy is an intracranial lesion the clinician must ascertain its seat, and in this respect the extent and the localization of the convulsions are of paramount importance. If the clinical distinction between an essential epilepsy and the symptomatic and reflex varieties cannot be made, it is equally clear that epilepsy may run a variable course, which is to be well borne in mind from a diagnostic point of view. We have seen what great interest in this respect attaches to the differentiation of the acute from the chronic forms of epilepsy. An equally great interest belongs to the diagnosis of the site of the lesion in distinguishing partial from general epilepsy of sudden onset.

It is but fair to state, however, that this distinction is in fact purely theoretical. Thus in the majority of cases of so-called general epilepsy we observe either a unilateral predominance or strictly localized initial convulsions; very rarely are the convulsions exactly symmetrical, and sometimes we find that attacks which at first were generalized gradually become more restricted and in the end are limited perhaps to a logospasm.[24] On the other hand, we may add that strictly localized lesions such as tumors have given rise to ordinary epilepsy.

With this understanding it is correct to say that the symptom complex called partial epilepsy, when well established, has a definite semeiological value; it signifies an irritative lesion localized in the brain on the side opposite to the convulsions. This symptom complex belongs not only to cerebral tumors but also to localized meningitis, encephalitis, patches of softening, etc., and may be presented by uræmia (Raymond, Tenneson and Chantemesse, Chauffard, and others). I shall not again point out the concomitant symptoms which enable us to make the diagnosis in different cases, and shall only dwell especially on points serving to permit an exact localization which is particularly necessary in the case of a cerebral tumor justifying surgical intervention.

Let us suppose that the diagnosis of an intracranial tumor has been correctly made. The convulsions are clearly partial and constantly occur in the same regions; atrophy of the papilla has been demonstrated by the ophthalmoscope; persistent fixed headache is present on the side opposite to that on which the convulsions appear, at the level of the so-called motor region of the cerebral cortex, etc. How is the seat of the lesion to be determined?

In the great majority of cases partial epilepsy is caused by a cortical lesion. When the epilepsy is hemiplegic from the start, that is to say, involves an entire half of the body, it would be reasonable to suppose that the lesion extends over the greater portion of the motor

centres. This supposition would be still more probable when a motor hemiplegia is present on the same side. But the spasm may remain localized, or at least may be so at the beginning of the attack before it spreads over the whole side. The localization of the lesion may be established by the following rules based on a certain number of facts verified by autopsies: 1. Convulsions commencing in the fingers or the upper extremity correspond to a lesion of the ascending frontal convolution of the opposite side. 2. A lesion situated on the summit of the ascending convolutions and encroaching on the fissure of Rolando induces convulsions in both the upper and lower extremity. 3. Convulsions starting in the face invariably coincide with lesions of the inferior portion of the ascending convolutions. 4. Those starting from the thigh are due to a lesion of the first frontal convolution. 5. Finally, lesions located towards the central portion of the ascending convolutions are indicated by convulsions of the face and of the extremities of the opposite side.

It ought to be known, however, that lesions of the cortical motor zone are not the only ones that may cause spasms; the latter may be observed also in consequence of lesions of the frontal or the parieto-occipital lobe; the former are often associated with disturbances of the intellect, the latter with disturbances of sensibility.

The sensory or sensorial phenomena of the aura may furnish a useful indication in diagnosing the site of the lesion, especially when the spasm is at once generalized over an entire half of the body. Thus we may suspect a lesion of the occipital or sphenoidal lobe when the convulsion is preceded by sensorial disturbances of vision or of the audition of words, etc.

Finally it may be remarked that cortical lesions are not the only ones which might excite partial epilepsy. All the lesions involving the pyramidal tract or the motor fibres of the tongue or the face may produce strictly local convulsions, whether they be situated at the level of the cortex, of the centrum ovale, of the corona radiata, or of the peduncle.

It is important to determine the nature of the lesion, especially if the question involves a tumor accessible to surgery. Tuberculous tumors are more frequent in young persons; syphilitic growths in individuals between twenty-five and sixty years of age. Moreover, syphilis is the cause of almost one-fourth of all the tumors affecting the brain (Ball and Krishaber). Finally, cancer is observed most frequently in advanced age, though this is not an invariable rule. When the tumor causes a perforation of the cranium, the probability of its cancerous nature is great.

Epilepsy may be simulated by certain persons with a definite ob-

ject. It is one of the most easily simulated diseases, since aside from the paroxysms it is compatible with otherwise good health. It is often very difficult to pronounce upon the genuineness of an attack which has not been witnessed. When the simulated attack takes place in the presence of an experienced physician who is familiar with the symptoms of epilepsy, the deceit will in the majority of cases be discovered by some circumstance. At the same time it must be admitted that there is no constant pathognomonic sign of an epileptic fit. Dilatation with insensibility of the pupil constitutes still one of the best signs of the genuineness of the attack, some statements to the contrary nowithstanding. The sphygmographic tracings of the radial pulse after the fit (Voisin) possess less importance than has been ascribed to them; I have obtained analogous tracings after prolonged muscular effort in normal subjects. It is obvious that the discovery of some trick such as the introduction of soap into the mouth with the intention of imitating salivation, etc., will unmask the malingerer. In true epileptics the simulation of an attack may produce a genuine one.

Prognosis.

Epilepsy is always a serious affection. Numerous accidents may occur at the moment of the seizure, such as a fall into the fire, fracture of the cranium or of the bones of the extremities, luxations, etc. Death may even be due to the fit, sometimes by asphyxia induced by a pre-existing lesion of the lung, sometimes by arrest of the heart or by a rupture due to some pathological state. Not only may the heart rupture during an attack, but also the liver and the diaphragm. In nocturnal attacks the patients are sometimes asphyxiated under their coverings or by pressure of the face against the pillow. When the fit occurs during a meal, death may be caused by the passage of food into the air passages. But it is mainly after serial crises, in the status epilepticus, that a fatal issue is observed. It is not rare also after eclamptic crises, and it may occur in consequence of a vertiginous status epilepticus.

The increased blood pressure may produce not only ecchymoses but large effusions into the cellular tissue or smaller exudations into the anterior chamber of the eye. The great violence of expiration may cause rupture of the frontal sinuses with resulting pneumatocele (Claus and Van der Stricht).

In a general way, partial epilepsy is less serious than general epilepsy. This statement, however, does not apply to epilepsy caused by cerebral tumors associated with symptoms of compression, in which the prognosis is particularly unfavorable. When the dis-

ease is due to syphilis, the results of treatment may furnish grounds for hope.

General epilepsy is more serious in proportion to the length of time it has existed, to the intensity of the attacks, and to the more prolonged stage of stertor. When the disease depends upon a well-marked peripheral lesion as an exciting cause, such as an affection of the ear or a visceral disturbance amenable to treatment, the prognosis is somewhat better. But it will be necessary to intervene early, before the nervous system has, as it were, got into the convulsive habit, otherwise the removal of the cause may produce no result. The same remark applies also to syphilitic and toxic (alcoholic and saturnine) epilepsy.

When epilepsy is associated with symptoms of chronic diffuse encephalitis, the prognosis becomes extremely unfavorable.

Epilepsy may be recovered from spontaneously; this is most frequently the case with the epilepsy of early infancy. A large number of children suffer, in connection with dentition and digestive disturbance, from convulsions which do not recur. Spontaneous recovery is also possible, though less common, in the epilepsy of adults.

The introduction of bromide of potassium into therapeutics has remarkably improved the prognosis. Absolute recovery persisting ten, fifteen, and twenty years is rare, but amelioration is frequent. Vertiginous epilepsy seems to be even more rebellious to treatment than psychical epilepsy.

The sequelæ include not only dementia resulting from the cerebral deterioration, but also, owing to the physical decline ultimately ensuing, tuberculosis, infectious diseases, pneumonia, typhoid fever, etc.

Morbid Anatomy.

Among the lesions to be found at the autopsy of epileptics, a certain number are the consequence of the disease. These are the cicatrices of wounds or burns presented by the skin, especially in the prominent regions, *e.g.*, in the face, the supraciliary regions, on the frontal and parietal eminences, the external occipital protuberance, the olecranon, and the acromion. Often we find consolidated fractures, and old luxations which have become irreducible.

When death has been the result of a fit, and particularly of the status epilepticus, we find an intense congestion of all the viscera and especially of the brain. The sinuses and veins are gorged with dark blood and the meninges are injected. The white substance of the brain presents a red stippling, sometimes with punctiform hemorrhages; the gray substance, a more or less dark pink color. The floor of the

fourth ventricle is rose gray, sometimes dotted with ecchymoses; the substance of the cerebellum presents a wine color. All these signs of congestion of the brain seem to be, as Magendie thought, the mechanical product of the convulsion.

Other lesions may be considered as the cause of the disease. It is not necessary here to describe all those which may play the part of exciting causes of epilepsy, but I shall restrict myself solely to the brain lesions of epileptics.

Great differences have been noted in the volume and weight of the brain of epileptics—microcephaly, excessive volume of the brain, and differences in the weight of the two hemispheres. In so-called essential epilepsy there have been described indurations of different portions of the brain and the medulla, especially of the olivary bodies, of the hippocampus, and of the cerebellum. These indurations have been observed on certain regions of the convolutions, in the motor zone, on the temporosphenoidal convolutions, in the island of Reil, etc. They show themselves in the form of patches with a grained appearance, slightly elastic, and comparable to crushed morocco leather or to the surface of a cauliflower. The meninges present no alteration at the level of these lesions. These indurations have been well studied histologically by Buchholtz, and especially by Chaslin. The latter has shown that they consist of a neuroglia sclerosis with integrity of the vessels and of the pia, and his theory is that they form an evolutionary lesion, in view of the entire absence of inflammation in the cases observed by him.

Bleuler also found analogous lesions. Blocq and Marinesco observed this sclerosis only exceptionally; in some cases they failed to find a similar lesion, more frequently they discovered in the psychomotor region some vascular lesions and lesions of the cells infiltrated with granulation. Marinesco and Sérieux[25] are "disposed to admit a modification of the protoplasm of the nerve cells."

Along with these patches of induration, Rilliet and Barthez, and more recently Bourneville and Brissaud, described a tubercular and hypertrophic sclerosis of the brain, in which the convolutions are covered with projections and tuberosities which may attain the volume of a large nut. These protrusions consist essentially of a sclerous tissue which may eventually destroy the nerve elements and the vessels.

Crocq has given an endarteritis of the basilar artery and its branches as the cause of epilepsy in the aged. Claus and Van der Stricht ascribe the more general rôle to an infectious endarteritis, but do not furnish sufficient proof.

General paralysis of the insane, which numbers epilepsy among

its most important symptoms, is characterized by a rapid degeneration of the cells and fibres of the gray cortex in its cerebral lesions.

We know that extremely varied brain lesions may cause epilepsy, *e.g.*, cerebral tumors of every kind, acute and chronic meningitis, aneurysms of the large arteries of the brain, meningeal hemorrhages, and hæmatoma of the dura.

The lesions of partial epilepsy of adults affect most frequently the cerebral cortex. They are generally situated in the cortical motor zone (Charcot and Pitres), but they may also be located outside of this zone; they then act by irritation from a distance. Consequently the topographical diagnosis of a cortical lesion productive of Jacksonian epilepsy cannot be made with certainty if the convulsions alone are considered. The paralyses in the form of permanent monoplegia or hemiplegia possess quite a different value from this point of view. They indicate a destructive lesion of the cortical motor zone of the opposite side.

The syphilitic lesions comprise principally arteritis and encephalitis or gummatous meningitis.

Pathological anatomy, aided by clinical observation, shows that these limited motor disturbances correspond with lesions localized in the cortex, according to pretty well-established rules. The reader is referred to what has been said above in connection with the topographical diagnosis of the lesions.

As to the sensorial localizations, although certain anatomical and clinical facts speak in favor of their being likewise situated in the cortex, we lack precise data for determining the laws governing them. In one case of sensorial discharge, consisting of flashes of red and white light in the right eye with temporary hemianopsia, Byrom Bramwell observed a coarse lesion limited to the posterior extremity of the occipital cornu of the left hemisphere.[26]

Epileptic dementia has long been considered as having no characteristic lesions. Bevan Lewis and Whitwell demonstrated the existence of a degeneration of the ganglion cells of the cerebral cortex, consisting in a nuclear vacuolization, sometimes, but not constantly, associated with pigmentation. This form of degeneration is not peculiar to epilepsy, for it is also met with in alcoholic dementia.

Pathogenesis.

For the first scientific pathogenic theory of epilepsy we are indebted to Marshall Hall. This theory located the disease in the medulla. Based as it was on the experiments of Claude Bernard, Brown-Séquard, Kussmaul, and others, and on the pathological re-

searches of Schroeder van der Kolk, this theory has found numerous advocates. Despite the various lesions discovered in post-mortem examinations, the disease was always attributed to the abnormal hyperexcitability of the medulla. This hyperexcitability, moreover, it was claimed, could be brought about either directly or in a reflex way through the influence of a distant cause. Germain Sée defined epilepsy as a disease characterized by an hereditary, congenital or acquired, but invariably permanent exaggeration of the reflex activities of the medulla oblongata.

While the medullary theory is able to account for the motor phenomena, for the respiratory and circulatory disturbances, it is insufficient in other respects, particularly to explain the psychical phenomena. The cortical theory of epilepsy, which is gradually gaining ground since the first observations of Hughlings Jackson (1863), is much more satisfactory in this respect.

It is now known, from the experiments of Fritsch, Hitzig, and Ferrier that the gray cortex of the convolutions is directly excitable, contrary to the belief formerly entertained. It is furthermore known that the cortical excitations of the same region give rise to constant reactions, that is to say, to muscular spasms which are always alike (Carville and Duret, François Franck and Pitres). Horsley and Beevor have most accurately determined these cerebral localizations in the monkey. In man, as we may convince ourselves, for instance, on trephining, the same effects are produced. Localized irritations of the cortex have excited convulsions in the face and in the limbs of the opposite side.

This cortical theory explains not only all the motor phenomena, but it alone can account for the psychical paroxysms presented by epileptics, for the emotional states described above, the intellectual auras, the abrupt modifications in the affective sentiments which usher in the attack, etc. The same interpretation harmonizes with other facts, such as the association with epilepsy of symptoms of aphasia and word blindness—phenomena whose cortical localization is well known.

The importance which attaches to the participation of inferior nerve centres, however, cannot be denied, were it only in the generalization of the convulsive phenomena. The excitability of the cortex is put in operation either directly or in a reflex way, under the influence of a peripheral irritation. The cortical centres give the signal for the explosion which reverberates in the inferior centres.

I still have to indicate briefly the pathogenesis of the principal phenomena of the attack. Like the latter, the aura results from a cortical excitation; it should therefore no longer be looked upon as a

peripheral cause of the fit, as was done formerly, but as forming an integral part of the attack, of which it is merely the initial phase. The loss of consciousness is easily explained, if we admit that the discharge has for its seat the superior cerebral centres, and it is not necessary to explain it by the intervention of cerebral anæmia according to the theory advocated by Brown-Séquard. The contraction of the peripheral vessels occurs at the onset of the fit and accounts for the pallor of the face and the increase of arterial tension.

But we must admit that loss of consciousness may ensue even after removal of the two superior cervical ganglia of the great sympathetic, as seems to be proved by the operations of Alexander. In a more recent paper[27] Brown-Séquard has been forced to the admission that the loss of consciousness is due to an inhibition, but this is no longer an explanation.

The theory of anæmia has still in its favor the experiments of Astley Cooper, of Kussmaul and Tenner, of Donders and Van der Beek Callenfels, of Nothnagel, and others. Salathé observed that when an animal is fastened to a platform in continual rotary motion, its brain becomes anæmic or congested, according to whether the head is directed towards the centre or towards the periphery; Gutnikow, who repeated the experiment with guinea-pigs after excision of the sciatic nerve, found that epileptic convulsions ensued only in anæmic conditions of the brain. Haig believes that an excess of urates in the blood causes contraction of the arterioles and capillaries; that this contraction leads to pallor and coldness of the extremities and may produce the same effects in the cerebral circulation, giving rise to epileptic attacks.

However, in absinthic epilepsy Magnan observed marked congestion of the hemispheres, and Todorski noted that during an attack produced by irritation of the cortex by means of interrupted currents the vessels of the pia dilated and the carotid pressure increased; and this occurred in the peripheral terminations as well as in the central. Nevertheless the theory of anæmia is still generally accepted and Hallager recently again wrote in its defence. This theory may be adapted to the cortical localization of epilepsy.

Since Fritsch and Hitzig demonstrated the excitability of the cortical substance of the brain, a large number of experiments have been made to determine the functional localizations. When the irritations of the motor region are limited and feeble, they induce monospasms or convulsions affecting only a small group of muscles; when the excitation is applied to a more extensive region, or when it is very intense or prolonged, the convulsive movements involve neighboring muscles or all the muscles of the extremity. When the excitation is

still more intense, it may affect one entire half of the body or even both sides. Ransom showed that electric irritation of the cerebral cortex in man caused movements preceded by a vague sensation in the parts involved. This sensation which is attributable to vascular movements (Schäfer) may be compared to the aura. Excitations of other than motor zones act only when they are very intense or greatly prolonged and their effect ceases after the motor zones have been removed (Pitres).

Irritation of the white substance of the convolutions may likewise cause convulsions (Bubnoff and Heidenhain). Ziehen also observed that excitation of the optic layer of the caudate nucleus may give rise to spasms. The experiments of Mott and Schäfer show that excitation of the fibres of the vertically divided corpus callosum causes contractions analogous to those produced by irritation of the motor zone of the opposite side; this helps us to understand how tumors of the corpus callosum provoke epilepsy (Ransom).

When an animal has previously undergone cortical excitation (François Franck), or when the cortical substance has been irritated by the application of some fluid like Liebig's extract (Koranyi and Tauszk), peripheral irritations determine in the muscles supplied by the irritated cortical territory convulsions which may become generalized when the irritation is intensified. The antecedent irritation gave rise to the formation of a *locus minoris resistentiæ* affected with an irritable weakness, and this helps to explain the part played by peripheral irritations in the causation of attacks in subjects suffering from congenital or acquired lesion of the brain.

The convulsions not only involve the muscles of the extremities, but those of the abdomen, the thorax, and the glottis are likewise affected. The initial cry has therefore been explained by the abrupt contraction of the thorax coinciding with the spasmodic narrowing of the glottis. The obstruction to the respiration and circulation accounts for the injection of the face which then follows, and also for the sanguineous effusions or hemorrhages which may occur. The incontinence of urine and fæces may be explained by the contraction of the abdominal muscles; but it has also been demonstrated that the bladder and the intestines contract independently during the convulsive stage.

The stertor and the clouding of the intellect and memory following the attack are to be explained by the exhaustion of the cerebral cortex consecutive to the discharge. For the interpretation of the post-epileptic paralyses we may avail ourselves of the theory of exhaustion formulated by Robertson and Tood and adopted by Hughlings Jackson. The non-excitability of the cortex after violent

repeated irritations has been experimentally demonstrated, and it is reasonable to suppose that the paralyses observed in the muscular regions which have been the site of violent convulsions are probably due to the exhaustion of cortical irritability by an intense discharge. Still Hallager explains it, like the attack, on the theory of anæmia.

The cortical centres give the signal for the discharge that reverberates in the medullary centres which explode in their turn; nevertheless, the existence of cortical centres is not indispensable to the production of partial or general epilepsy; a considerable number of anencephalous monsters die in convulsions. Goltz observed that dogs which had been rendered apathetic and stupid by the ablation of both hemispheres could still have convulsions; in one of Seppili's cases the centres were completely destroyed. According to Marinesco and Sérieux, bulbar lesions do not give rise to convulsions; the sensory, intellectual, and motor attacks would be explained by the rather special implication of the neurons of perception, of the neurons of association, or of the neurons of discharge.[28]

We know but little about the causes of the paroxysmal appearance of the seizures. Having ascertained by intravenous injections an increase in the toxicity of the urine before the paroxysm and a diminution after it, I have been inclined to attribute the discharge to an accumulation of toxic products in the blood.[29] Mairet and Bosc have obtained identical results.[30] Claus and Van der Stricht report analogous effects in operating by means of gastric injections. The researches of Herter and Smith are also favorable to the view of the toxic origin previously maintained by Flint. The latter author ascribed an important part to the products of intestinal putrefaction, and they have refuted Haig's theory that the attack is caused by the accumulation of uric acid in the blood; they deny in particular that there is a diminution of uric acid in the urine before the attack.

The coincidence of epileptic fits with peripheral neuritis (Cagney) may be cited in support of the toxic theory.

Treatment.

The treatment of epilepsy includes two important indications: on the one hand, the treatment of the attacks; and on the other hand the modification of the morbid constitution which gives rise to them, that is to say, the treatment of the underlying cause.

When the recurrence of the fits manifestly depends upon such a cause as alcoholism, moral emotions, or fright, we may hope to succeed in suppressing them or at least in lessening them considerably by the removal of this cause. For instance, the effect of abstinence is

one that can be clearly established in certain asylum patients who suffer from fits only after they have availed themselves of the permission to go out.

Attacks preceded by an aura may sometimes be prevented by the resort to measures which are largely empirical and are often employed by the patients themselves after they have learned by experience to recognize the significance of the premonitory signs. Thus when the aura consists of a peripheral sensation, such as a cramp in an extremity, the attack can often be arrested by a constriction of the limb above the seat of the sensation. Sometimes a strong ligature is required to obtain this result, sometimes constriction with the hand suffices.

Certain patients whose fits are preceded by a uniform mental representation may avoid the attack by concentrating their thoughts upon some other subject. Cases of this kind have been interpreted as a proof of the possible influence of volition upon epileptic discharges; this influence is indirect.

In other instances the manipulation to be performed for the prevention of the attack has no bearing upon the aura, and success is obtained by mere accident. One attack is arrested by compression of the supraorbital nerves at their points of emergence, another by the application of cold, by the ingestion of some cold liquid, by energetic flagellation, etc.

The inhalation of anæsthetics (chloroform, ether, nitrite of amyl, etc.) has been tried with the same object, but these agents, aside from the fact that their absorption is often too slow to produce a useful effect, may sometimes themselves provoke an attack.

When the convulsions have commenced, there is nothing, with rare exceptions, that can arrest them. The only measures to be taken are those of preventing harm to the patient, such as injury and suffocation.

When warned of the impending fall by the pallor of the face, he should be protected as much as possible from the shock, be placed horizontally upon his back, the head bent somewhat backwards, and he should be restrained in such a way that the violent and repeated movements may not result in contusions. The clothes should be loosened, especially around the neck. The windows are to be opened in order to lessen the difficulties of respiration as much as possible by a supply of pure air.

Certain precautions are indicated for the night. A considerable number of epileptics die of suffocation during their nocturnal attacks, being strangled either by their clothes, or by pressing their face against the pillow, or perhaps by being rolled up in the bed cover-

ings. Close supervision is therefore indispensable when nocturnal fits are anticipated.

Death by suffocation may also occur during the muscular resolution of the stage of stertor, owing to the dropping of the tongue upon the superior orifice of the larynx in the dorsal position. The head, therefore, should be inclined sideways so that the tongue may fall forwards by its own weight. Sometimes resort to artificial respiration becomes necessary.

The sleep which follows the fit should be left undisturbed. After an attack the patient should receive substantial nourishment in a most concentrated form.

During the attacks of psychical excitement I have obtained good results by physical agents whose effect is to lower arterial tension by causing marked dilatation of the cutaneous vessels. A mustard bath or wrapping in sheets dipped in mustard water produces so much relief in certain cases that the patients themselves demand the application when they feel the attack coming on. Bromide has not time to act and the narcotics, though they sometimes moderate the violence of the seizures, do not shorten them appreciably.

In the status epilepticus or during the serial crises we must begin by removing the patient from every source of excitement, by preventing all outside noises, and by darkening the room; and above all when the patient regains consciousness between the attacks he must be prevented from moving and speaking. Mechanical measures for lowering arterial pressure may prove useful (ligature of the inferior extremities, extensive sinapisms, etc.).

Various measures have been proposed for the status epilepticus. The bromides, even in large doses, are far from producing their usual effect in this condition. Little advantage has been derived from the employment of anæsthetics or inhalations. It is important to counteract the organic deterioration in this state by artificial alimentation. The patients, whose recovery I have witnessed after considerable elevation of temperature, were fed by the stomach tube during the entire duration of the status epilepticus.

In the acute epilepsy of pregnancy, puerperal eclampsia, abstraction of blood which is still often employed gives few encouraging results. The anæsthetics, especially chloral, succeed better.

When albuminuria has been demonstrated in a pregnant woman, a milk diet furnishes a good prophylactic measure against eclampsia. When the disease manifests itself at the end of pregnancy the induction of delivery may bring about the cessation of the attacks; in certain cases, however, they persist.

Before instituting general treatment in an epileptic, diligent search

The rule is always to resort to local measures in cases of this kind, for the treatment is usually effective. Sometimes insignificant irritations involving the terminal extremities of a nerve may be the cause of all the trouble and their removal may effect a cure.

In like manner the extraction of foreign bodies from the ear or the nasal fossæ, as well as the removal of nasopharyngeal polypi, has caused the disappearance of the epileptic crises. I have referred several times to auricular epilepsy. This demands appropriate treatment. In temporary obstruction of the Eustachian tube with compression of the labyrinth resulting from the vacuum in the tympanic cavity, insufflations of air through the tube are very effective in suppressing the vertiginous attacks which often complicate this affection. Anomalies of refraction must be corrected.

Epilepsy consecutive to cranial traumatisms furnishes important operative indications.

Partial epilepsy may appear in consequence of a slight trauma involving only the integuments. In similar cases, having thoroughly acquainted ourselves with the condition of the injury, we may resort to local revulsives which will suffice in the majority of instances. Still there are rebellious cases in which these measures fail, and in that event we should be justified in trephining. It may happen, moreover, that epilepsy is caused by an intracranial effusion of blood; symptoms of cerebral compression will then be present in addition and trephining may bring relief by allowing the blood to escape.

When the trauma is severe, as in fracture of the cranium and depression of the fragments, the indication is still more urgent. Epilepsy sometimes appears suddenly and fully developed under these circumstances and trephining is imperatively called for, so that any physician may be forced to perform it. It is evident that the site of the operation is plainly shown by the injury and hence the question of localization will not arise.

When epilepsy has developed a long time after the trauma, the correspondence of the probable localization of the disturbance with the position of the cicatrix forms an indication for operation. However, the statistics of the treatment by trephining of partial traumatic epilepsy should be revised and all cases excluded which have not been observed a sufficient length of time. Agnew has reported statistics of this kind in which there were only four recoveries among fifty-seven cases, while thirty-two others seemed to be temporarily improved. Judging from the facts given by this author, surgery ought to be particularly useful in primarily relieving traumatic depressions; but after the morbid habit is formed it becomes almost hopeless and medical treatment alone is effective.

Aside from traumatisms, in spontaneous affections like intracranial effusions and tumors operation holds out at least a hope of relief. Contemporary surgery can boast of several operative successes in the removal of cerebral neoplasms. In this intervention the question of localization presents great importance; the existence of local spasms coinciding with a fixed pain in the head on the opposite side, and particularly paralytic phenomena in the members affected with the spasm, furnish a precise indication of the road to be followed in the search for the neoplasm.

The statistics collected by Hale White and Allen Starr show that only two per cent. of all cerebral tumors justify a surgical operation; and in his critical study Kroenlein concludes, as Bergmann does likewise, that the indications for operation in cerebral tumors are very limited.

In the treatment of epilepsy of sudden onset, recourse is had to the trephine even in the absence of trauma and of any symptom of cerebral tumor, after all other therapeutic measures have failed. It must be admitted that the cures obtained under these conditions are doubtful, and it is reasonable to suppose that the improvement observed was not due to the trephining. In fact, it is well known that every violent intervention, whether medical or surgical, may temporarily modify the course of epilepsy.

While the methods of medication directed against the exciting causes of the disease have given encouraging results, those founded on theoretical conceptions and put in operation especially by the older physicians have proved futile. Thus blood-letting, which acts upon the congestion or plethora, has been employed in recent times with success by Lépine in the epilepsy occurring in certain large eaters of plethoric habit. Evacuants, purgatives, and emetics meet only very limited indications. Revulsives have scarcely given satisfactory results; flying blisters, however, seem to be of some service when the attack is preceded by a constant peripheral aura. Among the revulsives, likewise, slight ignipuncture applied repeatedly to the cranial region corresponding to the motor centres which seem to be the site of the discharge has appeared to me to be unquestionably effective in some cases.

Permanent compression of the carotids has been tried with a view to modify the cerebral circulation; these and the vertebral vessels have even been ligated. The results offer very little encouragement, and the same remark applies to the removal of the superior cervical ganglion (Alexander).

The treatment of epilepsy, while addressed to the cause when it is accessible, must be directed also against the symptoms with the view

of producing a favorable modification in the spasmogenic tendency of the nervous system. I shall not here enumerate all the historical remedies which I have reviewed in my book on epilepsy, but shall limit myself to the most interesting.

Antispasmodics have long occupied an important place in the treatment of epilepsy; some of them are still in use. Among these may be named camphor, opium and its derivatives, valerian, and belladonna. This last drug is recommended by Debreyne and by Trousseau, especially for the nocturnal incontinence of urine which is often an epileptic manifestation. It is given in the form of powders of the extract; and the active principle, atropine, in the form of the neutral sulphate or of the valerianate, is also employed. I have observed from the use of belladonna an apparent cure in a case of serious vertiginous epilepsy.

The so-called rational methods of treatment of essential epilepsy have produced so few results that empiricism has become rampant, and long would be the list of remedies recommended as specifics for the disease and which would deserve trial only after the failure of all drugs that have been tested.

Let me mention only silver nitrate, oxide of zinc vaunted by Gaubius and restored to favor by Herpine, phosphide of copper, ammoniacal sulphate of copper, etc. Antipyrin relieves the headache and migraine, but it seems to have no influence on the course of the fits. In exceptional cases borax gives good results, but it exposes the patient to quite a number of cutaneous, digestive, and especially renal disturbances the gravity of which I have pointed out elsewhere.[31]

Bromide of potassium, first used by Locock in 1851 in the treatment of epilepsy, is still the drug most generally employed; the therapeutic successes obtained from its use and which have never been denied justify the favor in which it is held. The sedative effect of bromide of potassium manifests itself in the nervous system as well as in the smooth and striated muscular system and consequently also in the circulatory apparatus. It acts not only by diminishing the excitability of the nervous centres but also by moderating congestions, whatever their seat and nature.

The bromide should be administered by the stomach; it must be pure, that is to say, it must be free from iodide and especially chloride of potassium, and from the sulphate and carbonate of potassium. When taken in the fasting condition it sometimes causes cramps of the stomach; taken too soon after a meal it often gives rise to digestive disturbances; it is best given at the beginning of the meals.

The dose varies according to age; the average dose for adults is 4 to 8 gm. (ʒ i.–ij.) daily, but some patients can take with advan-

tage 20 (ʒ v.) and even 30 gm. (℥ i.) or more. Children of from four to five years easily bear 2, 3, or 4 gm. (gr. xxx., xlv., or lx.); at the age of ten to fifteen years they can take doses almost as large as those of adults.

The bromide may be prescribed always in the same dose or else in graduated increasing and decreasing doses; in the latter case we begin, for example, with 4 gm. (ʒ i.) and add 1 gm. (gr. xv.) each week until 7 to 8 gm. (gr. cv.–cxx.) are reached and then go back to the initial dose (Charcot). It may also be prescribed in massive doses of 15, 20, and 25 gm. (ʒ iv., v., vi.) every two, three, or four days, but I prefer the continuous method. The tolerance for the drug of any individual can be determined only by experience. We may increase the dose by 1 gm. every month so long as it produces no symptoms of intoxication, until the therapeutic effects become evident. The limit of the increase is merely the limit of tolerance. The therapeutic effect may manifest itself only after very large doses.

The bromide should not be stopped in the course of an effective treatment under any pretext, except in the case of an acute adynamic disease in which it would be formally contraindicated. Even when the disease seems cured the drug should be continued; the bromide remains almost an article of diet for the recovered epileptic. When the fits have not recurred for a year or two, we may try a gradual reduction of the drug, but this must be done with great caution, since it is a fact that the recovery is never assured and it is possible that with a premature arrest of the treatment the disease will recur in an acute form, as the status epilepticus, and the patient may thus be forced to settle his old account. The sudden withdrawal of the bromide, even when it seems to produce no effect, is not without danger of the occurrence of the status epilepticus.

Although radical cure persisting for many years is rare, amelioration is the rule, there being a diminution either in the number or in the severity of the paroxysms, whatever form they present.

The bromide may occasion certain accidents which it is useful to know and which are designated as brominism. Sometimes it is a true intoxication which presents itself in either an acute or a chronic form.

Acute brominism manifests itself in two ways, by inebriety and by stupor. Bromine inebriety consists of a state of more or less marked exaltation, together with anorexia, redness of the tongue, headache, and extreme irritability which disappears on withdrawal of the drug. In other instances, after a very brief phase of excitement, the patient falls into a kind of comatose condition from which nothing can arouse him; respiration is slowed, the pulse becomes weak, and death would unquestionably ensue if there were no intervention. The with-

drawal of the drug does not suffice to remove the symptoms; rapid elimination by the intestines must be effected by the administration of drastic cathartics. Recovery then follows very speedily.

Chronic brominism is characterized by a state of general depression. The face is pale, the mucous membranes are dry and colorless, and there is gradual emaciation; the features become expressionless and betray hebetude with subsequent true stupor; the limbs become weak, the sensibility is blunted, memory and intelligence are greatly enfeebled; finally fever sets in, the respiratory passages are engorged, and the patient succumbs with symptoms of adynamic pneumonia. Timely intervention, as in the preceding case, may prevent this fatal termination. In some cases the bromine intoxication acts especially on the functions of nutrition, giving rise to a cachectic state with emaciation and general atony. The administration of tonics, iron, quinine, and hydrotherapy will remedy this condition.

The most frequent complications of the bromide treatment are those presented by the skin. They take the form either of a disseminated confluent acne, especially in the face, in the dorsal regions, on the arms and thighs; or else of indurations in the shape of oval tubercles or elongated patches developing in the thickness of the corium. These indurations may become confluent and ulcerate, causing extensive losses of substance surrounded by a violaceous zone of a very characteristic aspect. They are observed principally on the anterior surface of the extremities, though they may also be present on the forearm and even the face. Their distribution is sometimes symmetrical. In young children the bromine eruptions occasionally attain an enormous development.

In order to prevent the cutaneous manifestations of the bromide the skin must be maintained in an extremely cleanly condition by antiseptic washes; the administration of arsenical preparations is very useful, and finally elimination by the kidneys must be favored by giving diuretics or simple diluent drinks; milk is to be recommended. In a large number of cases intestinal antisepsis proves markedly efficient in counteracting the incidental effects of the bromide, whose action it even seems to aid. The utility of antiseptics enables us to understand the well-known good effects of purgatives administered empirically.

Bromization by large doses favors the development of certain infections, notably pneumonia; hence buccal antisepsis should not be neglected.

The bromide when exhibited during pregnancy does not hinder the development of the fœtus, nor has it any deleterious effect upon the puerperium. On the contrary, gestation constitutes an indication

for the administration of bromides to epileptics; even if the drug have no effect upon the mother, there is reason to believe that it exerts a favorable influence upon the product of conception. Several instances of brominism caused by lactation have been reported, hence nurslings may be acted upon by giving the drug to the wet-nurse.

Other preparations of bromine have been used in epilepsy. The bromides of sodium, lithium, and ammonium have given good results. The combination of several bromides seems to have produced even better results than the exhibition of any single bromide, but positive proof of this still remains to be furnished. Mention may also be made of the bromides of camphor, arsenic, calcium, zinc, and gold.

For several years I have experimented with bromide of strontium, which in the majority of cases seems to have been equally efficacious as the bromide of potassium, but less liable to cause gastric disturbance.

The bromides, especially that of potassium, have been frequently combined with other drugs such as belladonna, opium (Flechsig, Benneke), codeine, and adonis vernalis (Bechterew); but these combinations seem to have succeeded no better than the bromide alone.

As an adjuvant to the internal medication in epilepsy such physical agents as hydrotherapy and electricity are often employed with success.

Hydrotherapy, for whose methodical application in the treatment of epilepsy we are indebted to Fleury, seems unable to effect a cure by itself, but when associated with the bromide treatment it induces a favorable modification of the general condition.

Electricity is employed chiefly in the form of the continuous current. The practice includes galvanization of the sympathetic or of the head, the current being passed obliquely from the frontal region to a point diametrically opposite at the nucha. Althaus also recommends transverse galvanization of the mastoid apophyses. Fischer advises general faradization.

Some special symptoms are improved by a few very simple agents. The headache, which is sometimes so intense in epileptics that it has been proposed to relieve it by the application of the trephine, may be ameliorated by a much more gentle measure, by compression. Briquet, who recommends its efficacy in hysterical headache, advises permanent compression by means of a circular band. I have successfully used a very simple apparatus which permits of the exercise of uniform compression upon the entire head, or of local pressure upon some point. It consists of a cloth cap with double walls divided into radial compartments which may be filled with shot.

The treatment of epilepsy should properly be combined with some

measures of general hygiene. It is self-evident that certain professions or bodily exercises should be prohibited. The patients should not be allowed to expose themselves to the danger of fire, water, a fall from a height, etc. This rule applies also to intellectual labors which require great mental application, and to social conditions which demand continual preoccupation.

Open-air exercise is beneficial to epileptics, provided it be moderate, but violent exercises may induce an attack. The alimentation of these patients demands no special consideration except that it should be nourishing and very abundant, for a large number of them are debilitated, anæmic, scrofulous, or predisposed to tuberculosis. Of course, digestive disturbances must be attentively treated. The quality and quantity of the food should be constantly watched over, for any dyspeptic trouble is liable to cause paroxysms.

The education of epileptic children presents the greatest difficulties; their excessive impressionability, their abrupt changes of temper, and their impulses must be counteracted with extreme circumspection. In our intercourse with them they must become convinced both of the good-will of every one around them as well as of the supremacy of the law. Their moral education is even more difficult than their physical or intellectual instruction; here above all the necessity presents itself to adapt the punishment to the fault, and to inflict it immediately after its commission if we desire that it should be understood as punitive, and thus do good.

Young epileptics must be closely watched with reference to the sexual functions. The habit of masturbation which is frequent among them, and very baneful in their condition, may possibly depend upon some local irritation which should be removed. For instance, circumcision has been performed with success in cases of excessive length or narrowing of the prepuce. Even in adults, genital excitement of any kind generally has a deplorable influence upon the course of the disease. Therefore, the patients should be removed as far as possible from such excitement; marital intercourse should be almost absolutely interdicted.

The number of epileptics is sufficiently large to make their social condition a problem of the highest interest. The distinction between dangerous and harmless epileptics is not so easy to make as might be believed. It can never be asserted that a patient of this class, who has previously been quiet and inoffensive, may not become violent at any moment.

A large number of these patients, even when they are exempt from mental disturbances, are obliged to give up their profession on account of their convulsive fits. And even if they are able to assist in

their maintenance, they still become a charge on their family or the public. They would be sufficiently assisted if they were furnished simply the means of following their vocation at liberty in surroundings adapted to their infirmity and interested in tolerating it. Many would be benefited by the family plan which is practised in Belgium and Scotland. At all events they should be collected in hospitals and not in asylums. In practice a large number are nevertheless admitted to the latter; for being often unable to get assistance in hospitals they prefer, with the connivance of the family, to be treated as insane, rather than to remain without any help.

Epileptics who have frequent attacks or fits followed by mental disturbances and intellectual weakness, and those suffering from idiocy or imbecility, are properly cared for in insane asylums. In the large asylums in which such patients are numerous it is best to bring them together in a special section, for they are often quarrelsome and a source of annoyance to the other patients.

Violent epileptics should be committed to special institutions on the responsibility of those in authority. We may say that an epileptic is dangerous when he commits damage or is violent in his behavior, and is only prevented by force from doing injury. Against the former society may protect itself by placing him under conditions which will prevent him from doing damage thereafter, and these measures must be permanent, for there is no objective fact that would permit any one to affirm that an epileptic is cured. As to the latter class, when they are restrained by public authority, or on the request of their friends, this can be done only with the object of guarding against possible, but not proven danger. As the law cannot admit crime by thought, and as it cannot ascribe a criminal intent to a brutal act often committed without motive and without demonstrable premeditation, it would be permissible, when the disease has been modified and the patient no longer presents any disturbances calling for intervention, to set him at liberty if his family will become responsible for his good behavior.

The French criminal law says in article 64 of the penal code with reference to mental affections: "There can be neither crime nor offence when the accused was in a state of dementia at the time of the act, or when he was constrained by a force which he was powerless to resist."

The Society of Legal Medicine in 1875 terminated its discussion on the responsibility of epileptics by the statement "that the general rules governing the examination into the responsibility of the insane apply likewise to epilepsy."

This is not the place to discuss legal provisions, but we cannot pro-

test too earnestly, both in the name of science and in the name of social utility, against the legal inequality established in favor of the insane. I have elsewhere insisted upon the impossibility of making a general distinction between criminals and the insane, based upon objective characteristics. But, on the other hand, the law in my opinion cannot establish a distinction between the different categories of dangerous persons, and should be made to apply to all indiscriminately. When the accused has been convicted as the author of the crime the same verdict should be rendered against him, be he insane, passionate, or of sound mind. It is only in the carrying out of the sentence that account should be taken of the condition of sanity or disease of the convict, and an enlightened criminal administration may apply the measures best suited to improvement or cure in the individual case.

Be this as it may, the conduct of the forensic physician in a similar case should be extremely reserved; he must state only what he has himself ascertained, and he should not apply his observations to the text of the law. The question as to the equitable decision and the moral responsibility are out of his province. When the expert himself has been a witness of an attack and when he has determined in the accused certain somatic phenomena which are sufficiently characteristic, he may testify to the existence of epilepsy. But he must be more reserved with reference to mental disturbances which are not in themselves characteristic, as well as in the interpretation of acts which he has not witnessed; for the characteristics which are considered as specific in epileptic impulses—unconsciousness, amnesia, absence of remorse, etc.—are beyond control and should never enter into scientific expert testimony.

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THE SPASMODIC NEUROSES.

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THE SPASMODIC NEUROSES.

CHOREA.

Chorea Vulgaris.

ORDINARY chorea, the chorea of Sydenham and of Bouteille, although described as an independent morbid state, as a neurosis, has come to be considered, in the latter half of the present century, as a manifestation of a general condition, especially of acute articular rheumatism. Germain Sée, with some restrictions, admitted the existence of purely neurotic cases, but Roger looked upon it as always rheumatismal in character. Bouchut advocated the anæmic theory. Others interpreted chorea as a reflex phenomenon. Those who still defend its independence have classed it as a neurosis of growth in close relation with hysteria, as a cerebrospinal neurosis of evolution, dependent upon which are cardiac and rheumatic manifestations.

In my opinion chorea is a symptom complex which resembles hysteria both in its etiology and in its manifestations, the latter comprising motor, sensory, and intellectual disturbances. This symptom complex, whether complete or partial, manifests itself under various conditions. Its clinical picture closely resembles that caused by certain cerebral lesions, thus enabling us to draw legitimate deductions as to the localization in the brain of temporary or permanent disturbances of nutrition which constitute the underlying anatomical condition.

ETIOLOGY.

Chorea appears most frequently in childhood, especially between the sixth and fifteenth year, though it may be observed in the adult and even in old age. Senile chorea, however, is as exceptional as congenital chorea. Chorea is quite often hereditary, especially in its chronic form (Huntington); but much more frequently it is one of the neuropathic family taints, that is to say, the patients generally belong to nervous families and are nervous themselves. All debilitating conditions favor its development; as a rule it is most frequent in puny, anæmic, chlorotic, scrofulous, and tuberculous

children, and may be observed in consequence of faulty alimentation, bad hygiene, sexual excesses, masturbation, school drudgery, a painful emotion, or a hemorrhage; it is often preceded by troubled sleep which may be included among the etiological conditions as well as among the symptoms. Certain states of fatigue have a particular influence upon its production, for instance child-bearing. In chorea gravidarum it is especially difficult to distinguish the respective parts played by physical and moral conditions. Chorea appears most frequently during the first pregnancy, sometimes in the second, rarely in later gestations. Women who have chorea in the second pregnancy often suffered from it in the first. I know a woman who was choreic in all of her five pregnancies. Chorea of pregnancy often develops in women who had suffered from infantile chorea. Exceptionally chorea gravidarum may develop at the end of pregnancy, but more frequently during the first four months.

Acute articular rheumatism is the general disease in connection with which chorea occurs most frequently. G. Sée and H. Roger have chiefly contributed to the establishment of this relationship. But rheumatism is neither a necessary precursor nor an invariable concomitant of chorea, which may arise independently of it. Obvious or latent rheumatic infection seems to play merely the part of an exciting cause in the same way as the other infectious diseases, such as measles, variola, scarlatina, typhoid and intermittent fevers, cholera, blennorrhagia, influenza, pneumonia, whooping-cough, etc., not to speak of syphilis which has likewise, and justly, been given as a causative factor of chorea. Grasset and Rauzier' admit that chorea may be the sole manifestation of hereditary rheumatism, but the instance cited by them has reference to chronic rheumatism—an affection which should be separated from acute articular rheumatism and which resembles the neuropathies both in its origin and in its manifestations. Chorea may also develop in consequence of an intoxication; Demme's observation with reference to iodoform seems convincing.

Not only general physical or moral shocks may cause chorea, but it may be due also to local lesions or irritations such as intestinal worms, dentition, gastric disturbances, lesions of the nasal fossæ or of the pharynx, and traumatisms or surgical lesions of the extremities. Mention may likewise be made of ocular fatigue in connection with the efforts required by anomalies of refraction (Stevens, de Schweinitz).

The undisputed part played by moral emotions and by contagion is another fact confirmatory of the analogy between ordinary chorea and hysteria. It is known, moreover, that chorea often coincides with hysteria in young girls, that hysteria frequently simulates

chorea, and that a number of choreics present hysterical stigmata and particularly the ovarian (Marie).

Symptoms.

The invasion of chorea is often preceded by a more or less prolonged prodromal period, but we find in it the confirmed conditions of the morbid state. In this respect relapses are especially interesting because, before anything in his appearance strikes the observer, the patient feels himself attacked and gives notice of his condition. The initial phenomenon seems to be physical prostration. In a case of relapse, before any motor trouble was manifested and even before the patient had been able to recognize an increased irritability, there was marked fatigue with a lessened energy of the movements; the lowered dynamographic curve showed the step-like form seen in the fatigue of hysteria and the reaction time was prolonged. These facts were noted during the four days preceding the onset of the abnormal movements. In the prodromal period the attention is observed to diminish with the energy of voluntary motility; then morbid irritability develops, followed by motor and psychical instability. Modifications of the character and conduct are noted; the patient loses his gaiety, becomes anxious, capricious, irritable, hard to get along with; he seems indifferent to what concerns him the most; the weakness of the memory is striking. This condition often presents remarkable oscillations; under the influence of moral or physical excitations more or less prolonged phases of exaltation are observed; then when the circumstances change, especially in the evening, the patient passes from the most exalted joy to a profound depression. This prodromal period is sometimes marked by the appearance of dysæsthetic zones, particularly in the spinal region, by subjective sensations, such as numbness, prickling in the extremities, itching around the natural orifices, and photopsia. At the same time there are often insomnia, frightful dreams, or night terrors.

Mainly after physical or moral shocks we may observe a sudden onset marked by a convulsive epileptiform or hysteriform crisis, or by delirium, vomiting, or an intense headache, with or without febrile movement.

The convulsive movements constitute the most striking feature of the morbid state. Sydenham had remarked that in the beginning the movements consist in a weakness manifested chiefly in the legs by a kind of limp. It seems in fact that the initial phenomenon consists of a variable paresis of the muscles which no longer resist the action of their antagonists. We can demonstrate an instability which

often commences in a single member; if the right arm is affected the hand becomes incapable of retaining objects it has grasped, especially if they are small; the handwriting becomes irregular, then illegible, and finally altogether impossible. The arm, shoulders, face, and tongue become the seat of irregular twitchings which the patient is soon unable to conceal. These extend to the inferior extremity and to the other side of the body in a variable order of succession, but quite often the convulsive phenomena preserve a lateral predominance; exceptionally they remain limited to one side only.

The aspect of the patient is characteristic by reason of the discordance of the movements of the different muscles of the face and extremities. The forehead is wrinkled and smoothed again, the eyebrows are knitted or raised; the eyelids twitch, open, and close; the lips are stretched or lengthened; the labial commissures are drawn up and down with absolute incoherence; most frequently the homologous muscles relax on one side and contract on the other. The eyes participate in these movements, as does also the tongue, which is sometimes protruded from the mouth, and again clings to the palate, from which it is soon detached with a smack. The contortions of the features are associated with disturbances of speech; the articulation is hesitating or stuttering unless there is complete glossoplegia; the sounds are emitted by jerks and are often cut short by false intonations and noisy explosions. Singing becomes impossible because the patients are incapable of regulating and sustaining the expiration. The neck is bent to one side, stretched, and twisted. The movements of the limbs are no less disturbed; the arms are agitated, the shoulders are elevated, the hands drop such objects as they are holding or upset those they are trying to avoid. The gait is also peculiar; the choreic lifts his leg suddenly, throws it sideways, and sometimes falls. Mastication and deglutition are at times much interfered with. The digestive and respiratory functions are likewise disturbed by the movements of the thoracic muscles.

When the choreic movements manifested either during voluntary actions or in a state of repose are limited to one side, it is most often the left. Among 223 cases Sée observed 47 of hemichorea and 16 of partial chorea; but the localization is never so strict as in hemichorea or partial chorea associated with paralyses with gross lesions. It is rare for a partial spasm to remain limited to one group of muscles during the entire course of the chorea; this localization is at most a phenomenon of the onset or of the termination. Sometimes chorea presents itself in a crossed or alternating form, rarely it assumes a paraplegic type.

As a rule sleep interrupts the convulsions, barring grave excep-

tional cases in which they persist. The sleep is often of short duration and troubled by frightful dreams. Sleep induced by chloroform, chloral, etc., also interrupts the choreic spasms. Occasionally certain voluntary movements provoke a recrudescence of the spasms.

The pupils are often dilated and more so on the side subject to the severest spasms. Rosenthal and Benedikt have noted an increased electrical excitability. The tendon reflexes are generally exaggerated.

Sensibility in its various forms is found affected in nearly all cases in which it is carefully studied. Cutaneous sensibility is usually diminished, especially on the parts in which the spasms are most pronounced. In a considerable number of cases sensorial disturbances analogous to those of hysteria are observed. Painful phenomena also occur; sometimes there is a tegumentary dysæsthesia predominating in the regions where the convulsions are strongest, at other times there are headache, pains within the muscles or in the articulations at the level of the epiphyseal lines, neuralgia, chiefly in the course of the sciatic, and sometimes sensitiveness of the nerves to pressure.

As to trophic disturbances, little has been noted except loss of pigment in patches of the skin and hair, pigment spots on the skin, and alopecia areata. Otherwise the nutrition is but little affected; the temperature remains normal; and the urine, in which has been discovered an increase of urea (Bence Jones), urates, and phosphoric acid, with diminution of calcium, etc., presents no special characteristics.

With reference to the mental disturbances, which have been especially well studied by Marcé, we find a diminution of attention corresponding to the lessened motor energy and the perversion of the movements; a pronounced tendency to the perception of subjective sensations, hallucinations of sight in particular, sometimes of hearing, taste, smell, and tactile sensibility, rarely of the genital sense;[3] impairment of memory and of receptivity, and in consequence a change in character which becomes whimsical and irritable, while the conduct is often incoherent. The hallucinations are frequently a continuation of a dream or else they appear in the state between sleeping and waking. Exceptionally at the onset, but more commonly in the course of the chorea, we may observe a maniacal delirium, often with hallucinations. Sometimes the delirium is melancholic with a tendency to suicide. In cases of acute chorea recovery from the delirium is the rule; but in subjects with an hereditary taint choreic insanity may form the prelude to a precocious dementia or at least to a permanent intellectual degradation. Still, when a congenital taint favors

the development of mental disturbances in choreics we cannot say that these disturbances are exclusively of a degenerative nature and develop in connection with the chorea without being dependent upon motor troubles. The motor troubles of chorea necessarily induce disturbances of the attention and consequently of perception; they necessitate in a mechanical way intellectual and emotional troubles. The weakness and the anæmia (Hasse) may also occupy a place in the pathogeny of the mental disturbances of the choreics, as may the state of the heart. Acceleration of the pulse and palpitations are not rare in choreics who sometimes present some degree of arrhythmia; a kind of chorea of the heart has been admitted. The palpitations may coincide with a murmur at the base and may be dependent upon the anæmia or upon an organic lesion of the heart due to a rheumatic or other infection.

Aside from ordinary chorea, a spasmodic affection most common in adolescence, several other forms have been distinguished in accordance with special etiological features or with the symptomatic aspect.

The spasms may be lacking or few, at least during the greater portion of the course of mild or paralytic chorea described by Todd, West, Wilks, and Gowers, and specially studied by Ollive. This form is merely an exaggeration of the ordinary paresis of choreics. The paralysis may appear suddenly, independent of any convulsion; it may manifest itself after an attack of ordinary chorea, or simply a few days after the onset of the spasms which cease and reappear later. Moreover, the paralysis may affect all the extremities, one-half of the body, or a single limb; sometimes it is even limited to a group of muscles, for instance those of the neck. The paralysis is always flaccid without any tendency to contraction, but it is occasionally associated with rapid wasting, though without the reaction of degeneration. The tendon reflexes are generally abolished. The cutaneous sensibility is sometimes intact, but it is often diminished in the region deprived of movement. However, even when the paralysis seems complete it is not rare to see slight convulsive shocks agitate some one or more of the muscles.

The chorea of pregnant women or chorea gravidarum deserves special mention only on account of its serious character. It may manifest itself in the beginning of pregnancy or at any time during its course. Sometimes it is hereditary, at other times it is merely a relapse of a chorea of infancy or adolescence, and it is more frequent in primiparæ. It is marked by an intensity of the convulsive phenomena, sometimes accompanied by fever and dryness of the tongue; it is frequently complicated with delirium and occasionally terminates fatally; not rarely it causes abortion. Fortunately it is rather rare.[1]

Course and Termination.

Acute chorea as a rule lasts from one month to three or four months, but it may extend over a year and longer. Recovery is the ordinary termination. This happy issue is sometimes brought about by some general disease, by a trauma, or by a moral emotion. Generally it leaves no trace behind, but relapses are frequent and may recur four, five, six, or seven times at variable intervals; sometimes the attacks are, as it were, subintrant, or, to put it differently, we have to deal with simple remissions separated by multiple recrudescences.

A fatal termination is not frequent outside of pregnancy; it is hardly ever observed in subjects under seven years of age, and is most frequent between fifteen and twenty years. Death results most commonly from cardiac lesions, sometimes from a cerebral congestion or hemorrhage, at times from the violence and incessant recurrence of the choreic convulsions which prevent alimentation and sleep and lead to a progressive debility;[4] the convulsions end in collapse, and death ensues in the coma that often interrupts the convulsions.

Prognosis.

The prognosis of chorea, accordingly, is rather serious. Infantile chorea is generally more benign; after puberty grave cases are more frequent (Anstie). The absence of psychical disturbances indicates a lesser gravity. In nervous families chorea is often not so serious, but it is subject to relapses; frequently it is the prelude to a psychical deterioration. Sometimes the affection assumes a chronic character. Chronic progressive chorea, Huntington's chorea, the hereditary chorea of adults, may follow ordinary chorea, the chorea of Sydenham.

Chronic Progressive Chorea.

Etiology.

According to Charcot—and this opinion seems to me well founded—chronic hereditary chorea differs only in its substratum from Sydenham's chorea. Patients suffering from the chronic form are distinguished by more marked hereditary or family taints. The predominant etiological factor of chronic chorea is its family character and its heredity which is often direct and similar; Lannois cites a family seventeen members of which were similarly affected. Chronic chorea is rare in children; it attacks by preference adults and the aged; there is no difference as to sex. Infections, rheumatism in particular,

seem to have little influence upon its development, and it is also rarely due to pregnancy; moral emotions, on the contrary, play a prominent part in its etiology.

Symptoms.

When chronic chorea is not a sequel of ordinary chorea, which is the most common occurrence, its onset is slow and progressive. The motor disturbances often manifest themselves first of all in the face muscles, at other times in those of an extremity or of one side of the body. The movements are spasmodic, irregular, without rhythm, and are performed during repose as well as in connection with voluntary acts. These movements, which bear the greatest analogy to those of ordinary chorea, are, however, less frequent and less abrupt. Whether the chorea remains limited or whether it becomes generalized, the affected parts are in permanent agitation, which the patients often try to disguise by assuming attitudes which oppose the spasms or by executing useless but voluntary movements which more or less moderate the spasmodic movements.

In the face hardly any except the ocular muscles are exempt; the eyelids, lips, and tongue participate in the most varied contortions. The spasms of the tongue interfere considerably with speech, which is still more interrupted by laryngeal spasms often causing inspiratory and expiratory noises. The movements of the limbs and of the trunk resemble those of ordinary chorea; but their slowness gives to the attitude and the gait an aspect of drunkenness which has given rise to mistakes in diagnosis. The movements most frequently become generalized, but they may predominate on one side of the body or in the lower extremities.

The attempt has been made to ascribe a special character to the influence of the will upon the movements; but this influence is merely limited, temporary, and sometimes indirect in the sense that the suspension of the spasms is obtained only by the execution of voluntary movements. The emotions exaggerate the spasms, calm and repose moderate them, yet hardly anything but sleep suspends them.

Muscular power is diminished, but atrophy of the muscles is met with only exceptionally. The tendon reflexes are exaggerated; electric contractility, and the action of the sphincters are unaffected. General and special sensibility does not appear to be affected. There are no visceral disturbances. Under the name of *ballisme chronique* Massalongo has described a form of chronic chorea in which the amplitude of the movements recalls tarantism and hysterical choromania.

In place of the temporary mental disturbances noted in acute

chorea, we continually observe, with rare exceptions, progressive mental troubles; the memory and the intelligence become gradually enfeebled, and this condition may go so far as to constitute dementia. At first the intellectual deterioration is more or less conscient, the patients being subject to feelings and ideas of suicide which disappear during the progress of the dementia. The mental deterioration is sometimes interrupted by periods of irritability and excitement with hallucinations. Chronic chorea is serious by reason of the fatality of its progressive course, which terminates in death after ten, twenty, or thirty years in a state of cachexia and dementia, and also by the frequency of its hereditary transmission.

Congenital Chorea.

The etiological conditions as well as the symptomatic aspect justify the distinction of still another form, namely, congenital chorea which, however, does not always present a uniform picture.

Congenital chorea may present itself in the flaccid or in the spasmodic form.[a] Congenital non-spasmodic or flaccid chorea begins at birth. In the four authentic cases reported by Ballet, one of which was a personal observation, the abnormal movements appeared with the first days of life and began in the face and the upper extremities. As in the true chorea of Sydenham, the movements are conscious and involuntary, exaggerated by emotions and voluntary acts, and ceasing during sleep. They are irregular, disordered, and arrhythmical. Their amplitude is rather great. Finally, a very important characteristic is the absence of stiffness of the limbs or of muscular rigidity.

The cerebral and intellectual development of children thus affected is generally slow. Their intelligence remains below the average; they are backward, but they are not idiots. The cardiac lesions which are so common in infantile chorea are entirely absent.

As regards its course, congenital chorea is an essentially chronic affection, whose disappearance is not to be hoped for. As to the etiological conditions, all that an analysis of the cases reveals is that the two sexes are affected indifferently, and similar heredity has been established by Schlesinger and Rieder.

By the side of this first group of congenital flaccid chorea the author describes a second group said to be spasmodic. In these cases it is possible to demonstrate in connection with the choreic movements a certain degree of more or less pronounced rigidity, and in addition exaggeration of the tendon reflexes. This coincidence denotes a lesion of the brain of which the congenital chorea is a predominant or accessory manifestation.

These two forms of congenital chorea are clearly differentiated first of all by the symptoms:

In the former we can demonstrate neither muscular rigidity nor exaggeration of the reflexes, which are constant symptoms in the latter. Infants suffering from flaccid chorea are simply debilitated; those affected with spasmodic or diplegic chorea are idiots.

Moreover, from an etiological point of view, we may say that while in cases of cerebral diplegia labor was difficult and took place before term, at seven or eight months, in those of flaccid chorea, on the other hand, delivery was in all cases at full term and three times out of four it was normal and regular.

In a word, congenital chorea without spasm differs radically from symptomatic chorea and from cerebral diplegia.

As to the place to be assigned to the former in nosology, we may consider congenital flaccid chorea as belonging to the group of chronic choreas; the group which includes the tardy hereditary chorea of Huntington. There are, therefore, cases of hereditary chorea of precocious development, as there are those of tardy development. The time of appearance may perhaps be subordinate to the more or less pronounced taint transmitted.

Diagnosis.

The diagnosis of the different forms of chorea is not a very easy one, owing to their varieties, each of which may present difficulties of its own.

First of all, the choreic movements must be distinguished from *tremors*. While the former consist of ample arrhythmical and irregular spasms, the latter are regular and rhythmical oscillations of variable amplitude, but uniform in frequency in one and the same case. These characteristics permit the differentiation at first sight of chorea from the tremors of paralysis agitans, multiple sclerosis, etc.

Ataxic movements differ from chorea in that they do not occur when the patient is at rest and by becoming exaggerated when the eyes are closed. The oscillations of multiple sclerosis are also exaggerated during voluntary movements. Besides, these diseases develop at an age when chorea is rare and they are characterized by various troubles which distinguish them from chorea.

Hereditary ataxia with its ataxocerebellar gait, its dysarthria, and its intellectual condition may at first sight simulate chorea, but may be differentiated by the abolition of the tendon reflexes, by the nystagmus, etc.

The movements of the affection known as *convulsive tics* are systematized, do not usually manifest themselves during voluntary move-

ments, and are often accompanied by spasmodic exclamations, which are likewise systematized, and by fixed ideas. Under the penalty of a more or less painful distress the patients are able to suppress their movements for some time.

The spasms of *paramyoclonus multiplex* are generally provoked by peripheral excitations and may became tetanic; they usually spare the face. The movements of the fibrillary chorea of Morvan present the same characters and likewise cease during the execution of voluntary movements, but instead of affecting the whole of the muscle they implicate only a certain number of fibres or bundles.

Under the name of *electric chorea*, Dubini has described a febrile affection characterized by strong intermittent contractions accompanied by convulsive attacks which often leave behind paralyses with deviation of the face and usually terminate in death, it may be in a few days or after several months. It is probably due to an infection (Grocco) tending to a cerebrospinal localization; in fact the post-mortem examination frequently shows diffuse congestive lesions of the meninges and of the cord.

The *electric chorea of Bergeron* or electrolepsy differs from the preceding by its benignancy and by the character of the spasms which implicate the neck in particular and are rhythmical and brusk. This affection resembles hysterical chorea both in its form and in its rapid curability. Sometimes, moreover, rhythmical hysterical chorea follows ordinary chorea.

Double athetosis, which is a disease of childhood but has also been observed in adolescents and even in adults, is characterized by voluntary, slow, and undulating movements that manifest themselves equally during the voluntary acts which exaggerate them and in their intervals, sometimes persisting during sleep. These movements invade the face and the trunk, as well as the limbs which are affected especially in their peripheral segments; the fingers are agitated by particularly characteristic undulations. These movements are associated with permanent stiffness and sometimes with an increase in the volume of the limbs. Athetosic patients often have convulsive attacks and intellectual disturbances. Finally, though double athetosis is regarded as connected with cerebral lesions, cerebral sclerosis, arrest of development of the pyramidal tract, pachymeningitis, and cerebral atrophy, there are cases in which a careful autopsy fails to discover any lesion, and it could be left in the list of neuroses with equal right as chorea. The analogy is emphasized by the fact that hemiathetosis and hemichorea may be connected with cerebral lesions having the same location and may be accompanied by paralysis and sensory-sensorial anæsthesia of the same side.

Hysteria may imitate chorea in its paroxysmal manifestations, which, however, generally present movements having a much greater amplitude. Hysterical chorea oftener assumes a rhythmical form which is absolutely characteristic—malleatory, saltatory, and rotatory chorea—in which the patient executes systematic movements recalling some professional act such as striking with a hammer or some other more or less complicated motion.

Under the name of *fibrillary chorea* Morvan has described a myoclonic affection characterized by fibrillary contractions which appear in the beginning in the muscles of the calf and the posterior portion of the thigh and may subsequently extend to the muscles of the trunk and even of the superior extremities, but always spare the neck and face muscles. This affection, according to the admission of the author who described it, hardly differs from paramyoclonus multiplex of Friedreich, which consists of convulsive clonic shocks agitating a certain number of symmetrical muscles of the extremities and sparing those of the face. The frequency of the contractions of paramyoclonus varies from ten to fifty per minute and their succession is sometimes so rapid that the affected muscle is to a certain extent tetanized. Their frequency and the intensity vary also from one moment to another, as does their rhythm. Most frequently these spasmodic movements cause no displacement of the limb; they cease during sleep, but are almost uninterrupted during waking hours. They are moderated by voluntary movements, but are exaggerated by cutaneous irritations and especially by cold, by attitudes which tend to relax the affected muscles, or by compression of the latter. In paramyoclonus multiplex the muscular power, the electric excitability of the nerves, and sensibility in all its forms remain normal, but the reflex excitability of the tendons and the skin is generally exaggerated. Still, this description of Friedreich does not apply to all cases of paramyoclonus; sometimes the facial muscles participate in the clonic convulsions (Homen, Allen Starr, Francotte, Bechterew, Seeligmüller, and others); in some cases the convulsions produce a displacement of the limbs (Marie, Lemoine and Lemaire, Bechterew, and others); in other instances they persist during sleep; the spasms are often more intense on one side than the other, at times they are even limited to one half of the body; and finally the clonic shocks of paramyoclonus sometimes coincide with tremors, athetosic movements, and disordered movements which may be connected with chorea. Besides, Lemoine has described a case of paramyoclonus multiplex followed by psychical disturbances and by echolalia, recalling Charcot's convulsive tics. In a case reported by Raymond the paramyoclonus multiplex was associated with an ordinary tremor,

with choreic movements, and with signs of mental degeneration. Another case of the same author had begun with non-painful tics of the face. Paramyoclonus resembles the non-painful tics, convulsive tics, and electric chorea; but its principal characters which I have just described show sufficiently that it can hardly be confounded with ordinary chorea.

There is a special form of chorea that is particularly apt to be confounded with the paralytic manifestations of hysteria, namely, mild or *paralytic chorea* which has been well described by Todd and subsequently studied by West, Wilks, Gowers, Ollive, and Charcot, and which is characterized by the exaggeration of the paretic state habitually present in chorea. This more or less marked paresis may extend to a variable number of muscles.

The paralysis runs a different course in different cases. Sometimes it appears suddenly, independent of any spasm, and is heralded solely by a peculiar mental state, changes of character, a certain torpor, and some awkwardness; at other times, in the course of an ordinary chorea which has manifested itself normally for two, three, four, or more days, the paralysis takes the place of the disordered movements; again, in other cases, the chorea appears to run a normal course, the movements moderate, and recovery seems at hand, when the paralysis sets in. Finally, after certain intense choreas in which the spasms are incessant we observe the supervention of fever, anorexia, a pronounced saburral state, and adynamia; the paralysis which then follows and becomes general is often a precursory sign of a fatal termination.

The paralysis may implicate all the extremities, one half of the body, or a single member; in other instances it is limited to the muscles of the neck, and the head having lost its support drops forwards or backwards. From time to time we may note in the paralyzed parts the occurrence of indistinct choreic spasms, and the tongue, which is generally spared by the paralysis, continues to be agitated by spasms. Choreic paralysis is never followed by contracture but remains flaccid; as a rule it is not associated with an anæsthesia as marked as that accompanying hysterical paralysis, but often enough the sensibility is affected. Exceptionally we observe incontinence of urine and fæces. The tendon reflexes are generally abolished. The reaction of degeneration is not observed, but sometimes a rapid atrophy supervenes, which has been explained by a mechanism analogous to that given in explanation of the atrophies of articular origin, in view of the frequency of articular lesions in choreic patients (Rondot).

Prognosis.

The prognosis of chorea is very variable. It is less grave before puberty than later (Anstie). There is a certain relation between the intensity of the phenomena and their persistence; the slight cases as a rule run a shorter course and recover definitively, while the severer cases last longer and relapse easily. In the intense cases without nocturnal remission the prognosis is serious, not only as regards the duration but as to life. When the choreic spasms are associated with marked mental disturbances the outlook is still more serious.

Chorea gravidarum is as a rule remarkable in the intensity of the convulsive phenomena; it is especially grave since it frequently causes abortion and terminates fatally in about one-third of the cases. Even the lightest attacks of chorea are an evidence of a neuropathic taint and constitute a menace for the future. Mild or paralytic chorea is recovered from as readily as spasmodic chorea, excepting the cases in which it follows an intense chorea and is an expression of profound exhaustion. The chronic forms, whether congenital or not, are always serious, since there is no treatment that influences their fatal course. In the same way as the emotions and moral shocks exert an unquestionable effect upon the development of chorea, they are no less active in causing relapses and exacerbations. Marshall Hall observed long ago that choreic movements which had been suspended during sleep may reappear in dreams; the nature of the sleep therefore constitutes an element in the prognosis as bearing upon the course of the chorea.

Pathology.

Chorea is a symptom complex which manifests itself under very different conditions, and its pathogeny is intricate.

The anatomical history of pre- or post-hemiplegic chorea which usually coincides with sensory hemianæsthesia shows that a lesion of the posterior third of the internal capsule is capable of inducing choreic phenomena. There are sufficiently good reasons for the belief that these lesions act merely by irritating at a distance the motor elements and that this irritation may be realized at other points of the centrifugal pathway. The English school holds that the majority of ordinary choreas have their origin in cerebral lesions. Kirkes connected chorea with endocarditis and Broadbent maintained that there are frequent lesions in the corpus striatum and the optic thalamas due to embolisms derived from the diseased heart. A certain number of autopsies reported by Tuckwell, Gray, Fox, and others testify in

favor of this theory, and they agree with those on which the localization of hemichorea is based. This agreement is the more interesting in view of the facts that ordinary chorea often predominates on one side, that it is associated with a notable paresis, and that disturbances of sensibility are not always absent. More frequently, however, diffuse lesions of the brain have been observed.

Other parts of the nervous system than the brain have been found affected in chorea. Compression of the medulla oblongata by an overgrown odontoid apophysis (Froriep) and a hemorrhage into the central canal of the spinal cord (Steiner) have been recorded. Sometimes the lesions even involved the whole extent of the nervous system —brain, cord, and nerves (Elischer)—and Frey found the lesions of polyneuritis. The investigations made in the case of choreic dogs do not possess the interest which has been ascribed to them, since it is not well established that the chorea of the dog is identical with that of man. Still it may be stated that with the capillary embolisms experimentally produced by Angel Money, those in the brain caused merely irregular movements, while the medullary embolisms gave rise to marked choreic movements.

On the whole, the pathological anatomy of chorea remains obscure. Raymond draws the following conclusions from a study of seventy-nine autopsies: 1. In persons who succumb in the course of a simple chorea lesions of the brain and of the heart are most common; 2. Lesions confined to the encephalon are rare; most frequently the cord and the heart are affected at the same time; 3. Hyperæmia is especially frequent, then follows softening, then chronic encephalitis; 4. The mode of origin of the softening is often readily established; nearly always deposits are present on the valves, several times even emboli have been discovered in the cephalic vessels; 5. With the exception of this last lesion hardly anything has been described that could be compared to the findings in symptomatic choreiform states. The most probable hypothesis, however, relative to the localization of the lesion in ordinary chorea is to place it in the course of the pyramidal bundle, whose irritation may produce various spasmodic movements.

We have seen that rheumatism may account for a certain number of the lesions and that we might be justified in attributing to it other lesions, but rheumatism is not always a factor, being sometimes absent. Duchanteau,* in the compilation of various statistics comprising 1,600 cases, finds a proportion of 28.1 per cent. of rheumatic chorea and 71.9 per cent. of chorea exempt from rheumatism.

It had been formerly noted that chorea may manifest itself in consequence of general diseases which have since been included among

the infections. Strümpell in particular has called attention to the part played by infection in the pathogeny of diseases of the nervous system, nor has he remained long the only advocate of this view. Leredde has demonstrated that we may observe in choreic patients an infectious endocarditis independent of rheumatism. Pianèse (1891) described a special bacillus which he believed to be the cause of chorea. Since then Möbius, Berkley, Osler, and Dana have developed the theory of the chorea of infection, based partly on the clinical, partly on the anatomical facts. Triboulet[1] has laid particular stress upon this subject; he does not believe in specific infection like Pianèse, but in a variable infection which he has been able to demonstrate in several cases. In connection with a case of chorea observed in a girl suffering from tuberculous adenitis Massalongo stated that chorea may be explained by the action upon the nervous system of poisons formed in tuberculous foci. Dana found in a case of ordinary Sydenham's chorea diplococci in the meninges and in the cortical substance. Meyer reported a case in which the blood and different viscera contained staphylococci and streptococci which indicate the relationship of chorea to rheumatism. The agents which give rise to infections, intoxications, and autointoxications may also reproduce them. The influence of nervous, traumatic, or emotional shocks upon chorea might find an explanation, under certain circumstances, in the fact that they place the nervous system in a state of lessened resistance, thus favoring infection.[2]

Nevertheless, chorea is far from appearing invariably under the same conditions of rheumatic infection, intoxication, or nervous shocks; its development, therefore, depends upon a neuropathic predisposition springing from a morbid heredity or from a transformed heredity due to an evolutionary nervous disturbance at any developmental period. The predisposition shows itself more markedly at the most active evolutionary periods, at puberty and during the puerperal life.

Treatment.

As the pathogeny is very complex, the therapeutic indications are very variable, and the specific treatment of the disease still remains a chimera as in the time of Bouteille. Among the factors entering into the production of chorea we have noted a nervous temperament, an infection, or a shock which places the system temporarily below par; chorea is usually associated with a condition of general debility and with disturbed sleep. It is upon these elements that we must try to act.

When the chorea has developed under the influence of a local irritation, of a general condition, of an infection, or of a shock, the first efforts must be directed against the etiological factor—the suppression of possible causes of irritation such as intestinal worms, constipation, dental lesions, phimosis, ocular disturbances; rheumatic infection, whether puerperal or otherwise, should be met by appropriate remedies, and the nutrition should be improved by a tonic regimen. The treatment of the shock is not without some chances of success. Thus, in a young girl who had already had two attacks of chorea of respectively six weeks and two months' duration, one following scarlatina, the other influenza, I have seen a third attack which had supervened after a fall arrested in four days under the influence of repeated injections of artificial serum. A treatment of this kind may be useful by acting both on the mind and on the nutrition.

When the chorea is associated with debility marked by anæmia, general treatment is the best antispasmodic. The use of arsenic in the treatment of chorea dates back more than a century and still enjoys a well-merited reputation. It has been especially recommended for chlorotic and lymphatic patients; Ollive advised its employment particularly in mild chorea. Ziemssen and Seguin hold, with good reason, that if arsenic does not succeed more frequently it is because it is used in insufficient doses; in their opinion massive doses are less dangerous than small doses long continued.

Arsenic may be prescribed in different forms—arsenious acid, arseniate of sodium, and arsenite of potassium. Arsenious acid may be ordered for children of from eight to ten years in doses of 2 mgm. (gr. $\frac{1}{30}$), increased by 2 mgm. daily; but often it causes nausea and vomiting. Arsenite of potassium is prescribed in the form of Fowler's solution which contains one per cent. by weight of arsenious acid. Fowler's solution is given in doses of gtt. v. to viij. a day for children and gtt. x. to xx. for adults. According to Seguin it may be ordered in increasing doses up to gtt. xv., xx., and xxv. three times a day; the toleration of the drug will be facilitated by diluting the solution with a tumberful of carbonated water, alkaline or acid, which is to be taken in two or three portions during the hour following the meals. When Fowler's solution is very badly borne by the stomach it may be administered hypodermically (Riedcliff, Eulenburg, Widerhofer, Frühwald); the pain of the injections may be diminished by substituting cherry-laurel water for the water of the ordinary solution. The amount of the first injection is $\frac{1}{30}$ of a cubic centimetre and this is increased by an equal quantity every day.

The arseniate of sodium, often prescribed in the form of Pearson's solution, should be given in rapidly increasing doses, 2, 4, or

6 mgm. (gr. $\frac{1}{30}$, $\frac{1}{16}$, $\frac{1}{10}$) daily sufficing for slight cases in children. In severe cases we may commence with 5 mgm. (gr. $\frac{1}{12}$) daily and increase the quantity by 5 mgm. every day so as to reach 25 or 30 mgm. in five days (Cadet de Gassicourt); after continuing its maximum dose for three days, the quantity is again diminished by 5 mgm. every day. The drug is better borne in divided doses not to exceed 3 mgm. each. When this treatment fails after twenty days it should be discontinued; otherwise it may be kept up several days after the cessation of the movements. The arsenical treatment may cause not only gastrointestinal disturbances, but also cutaneous lesions, erythemas, bronze-colored pigmentation, especially at the articular folds, and paralyses due to peripheral neuritis.

Along with arsenic other tonic drugs have found application in the treatment of chorea. Iron in particular is very useful in anæmic subjects, and it is always an advisable adjuvant. Cinchona in its different forms may likewise be of service, and cod-liver oil, phosphate of lime, etc., may be given with success.

Sleep is an accumulator of nerve force, says Lasègue, and hence it is not surprising that it has been proposed as a means of treatment in a large number of affections dependent upon nervous exhaustion; in such diseases it constitutes indeed a curative agent of the first order. It should be recommended in the forms of chorea in which the irregular movements disappear during sleep. Bastian has kept choreic patients who were seriously affected, for weeks in a sleep which was interrupted only for meals. Prolonged sleep is all the more indicated since insomnia plays in the production of the attack a part which is more pronounced in proportion to its persistence.

In order to secure it, chloroform or ether are to be inhaled twice a day to the point of resolution. Nitrite of amyl has likewise been recommended for inhalation in doses of from three to ten drops three times a day (Ziegler); but these procedures are justified only in urgent cases. The most commonly used hypnotic agents in chorea are chloral, sulphonal, paraldehyde, and chlorolose.

Chloral was recommended more than twenty-five years ago by Gairdner in England; Bouchut advocated its use in France some years later. There is no question that the sleep induced by chloral causes a cessation of the agitation, but this circumstance alone does not demonstrate the utility of chloral in chorea. In general the statistics relative to the duration of the affection prove little; but one case reported by Gairdner seems to offer pretty strong evidence: one little patient took by mistake 4 gm. (ʒ i.) of chloral instead of 1.30 gm. (gr. xx.); she presented symptoms of intoxication but the chorea ceased. The object to be attained is prolonged sleep and thereby a

suspension of the movements; to this end we must commence with 1 gm. (gr. xv.) of chloral and increase the dose by 50 cgm. (gr. viiss.) daily; the drug being stopped only when depression of the pulse or symptoms of gastric irritation supervene. It should not be forgotten that chloral is always dangerous in subjects suffering from heart lesions. Often when sleep for from twelve to sixteen hours a day has been obtained the movements moderate and cure results; but we cannot disguise the fact that in a fair number of cases this cure does not occur until the chorea has run its usual course; and finally it is not rare that the movements, though calmed while the prolonged sleep is maintained, regain their activity the moment it ceases.

The same remarks apply to paraldehyde and to sulphonal. Paraldehyde acts both as a hypnotic and as a calmative agent upon the reflex actions of the cord; it may be given in doses of from 2 to 4 gm. (ʒ ss.–i.) daily. It has the advantage over chloral of not depressing the heart. Sulphonal may be ordered in wafers or suppositories in doses of from 1 to 2 gm. (gr. xv.–xxx.) a day.

Chlorolose, which has a special hypnotic effect upon the brain, while leaving the cord awake (Richet), is very suitable for the production of prolonged sleep, and I have used it with success in chorea;[9] but its suspensory action upon the cerebral functions may, in hysterical patients, produce paralytic phenomena[10] which should lead to some reserve in view of the possibility of causing paralytic accidents in chorea.

Opium in massive doses has been recommended by Jaccoud, who advises the induction of somnolence in young children; this method is dangerous and has found few advocates.

Antispasmodics, and first among them the bromides, have kept an important place in the treatment of chorea; if they have not shown the good effects in the latter which they have produced in epilepsy it may be because they have not been prescribed with the same conviction. To give the bromides in doses of 8 gm. (ʒ ij.) daily is a practice which seems to reach the limits of prudence. I admit that in my trials with the drug the bromide failed, but these experiments have not been numerous because I prefer to address my treatment to the general condition rather than to the spasms. It should be added, moreover, that the bromide of potassium has given good results in chorea gravidarum (Gubler, Jaccoud, and others).

Since the publication of a paper by Wallner, of Munich, in 1887, antipyrin has enjoyed considerable favor in the treatment of chorea. Some rapid cures, in a few days, have been reported with this drug, and its effects seem to be generally favorable; but some altogether refractory cases have also been published. It is prescribed in doses

of 50 cgm. (gr. viiss.), increased by the same quantity every day; the amount may go as high as 8 gm. (ʒ ij.) daily (Moncorvo). Even in children tolerance is maintained for weeks. Intolerance manifests itself by digestive disturbances, nausea, vomiting, diarrhœa, or skin eruptions; when given in large and long-continued doses the urine must be watched; in epileptics I have several times observed the appearance of albuminuria which ceased with the suspension of the treatment.

Exalgin seems to produce analogous effects to antipyrin (Moncorvo). Loewenthal and Dana have especially praised its efficacy. It acts like antipyrin in doses of half the size and is well borne by the stomach. It is prescribed in doses of 25 cgm. (gr. iv.) and increased by the same amount daily; children of from six to seven years easily bear doses of 1 gm. (gr. xv.) a day.

The valerianate and other salts of zinc are also sometimes employed. Recently the bromovalerianate of zinc has been recommended in doses of 0.50–1.50 gm. (gr. viij.–xxiv.).

Belladonna and atropine likewise have their advocates. The former has sometimes been associated with valerian and asafetida.

Bouchut advises the administration of sulphate of eserine in doses of from 2 to 5 mgm. (gr. $\frac{1}{30}$–$\frac{1}{12}$), but the marvellous results reported by him have not been substantiated in the hands of Riess, Gubler, and Cadet de Gassicourt. This drug, moreover, may occasion grave accidents.

Hyosycamine in doses of from 2 to 10 mgm. (gr. $\frac{1}{32}$–$\frac{1}{6}$) has given good results in Oulmont's hands. Magnan advised for grave cases hydrochlorate of hyoscine by subcutaneous injection in doses of from 1 to 2 mgm. (gr. $\frac{1}{64}$–$\frac{1}{32}$) for adults.

Among the drugs which have been employed in chorea as alteratives strychnine should be first enumerated, which Trousseau gave in the form of a syrup containing 0.05 in 100 (gr. ¾ in ʒ iij.) of the sulphate, in doses of from ½ to 6 cgm. (♏ $\frac{1}{12}$–i.) a day. The object was, it seems, to substitute tetanic contractions for the clonic movements. Although Hammond and Benedikt have reported facts tending to confirm Trousseau's confidence in the drug, strychnine remains under suspicion. Picrotoxin, which has been proposed for the same purpose, has not proved useful.

Tartar emetic, which was employed by Laennec and especially recommended by Gillette and Bouley, is hardly used nowadays. The advocates of the infectious theory might explain the success which has been attributed to it by its evacuant action; the advocates of the nervous theory would be no less justified in maintaining that it acts by producing a shock. Bouley's mode of administration is particularly

brutal: he gave the first day 50 cgm. and the next day 1 gm. in two doses at an hour's interval; if no improvement was produced he raised the dose to 1.50 gm. the third day; by this means he succeeded in bringing the patient into collapse. Gillette contented himself with progressive doses of 20, 40, and 60 cgm.

The external agents utilized in chorea are revulsives, electricity, hydrotherapy, gymnastics, and massage. As revulsives, have been employed light ignipuncture along the vertebral column, tincture of iodine, and atomizations of ether and methyl chloride. Electricity has been used in all its forms, but aside from the tonic action which static electricity may possess the effects are doubtful. Hydrotherapy likewise is mainly to be recommended as a tonic. The cold baths by immersion which were successful in Dupuytren's hands seem to act chiefly by shock. The most useful modes of application of hydrotherapy are those recommended for hysteria (see p. 570). Sulphur baths (Baudelocque) and douches (Cadet de Gassicourt) have sometimes given good results. In convalescence these baths may prove useful.

Regulation of the voluntary movements by gymnastic exercises, was long ago (1827) recommended in France for the treatment of chorea. Louvet-Lamarre, Blache, and Jolly advised exercises of precision, jumping the rope, dancing, and piano playing; but it was Laisné in particular who made a systematic application of gymnastics at the hospital for sick children, beginning in 1847. Germain Sée, Blache, and Bouvier recognized the utility of the method. In order to re-establish the influence of the will upon the movements it is necessary to have recourse to simple exercises in which the patient executes with precision movements which he sees performed before him and which are exactly explained to him. The most useful movements are those which are performed with an exact knowledge of their direction, extent, energy, and rapidity. The patients profit greatly by exercising before a mirror which permits them to regulate their movements with exactitude. The exercises should include the different qualities of movement—exercises of direction, placing the hand or the foot or the finger upon a given point; exercises of force, making a given traction on an apparatus with springs; exercises of rapidity, carrying a finger to one or several points, one or more times, in a given time, etc. The exercises of the limbs should be combined with exercises of respiration whose regularity serves as a point of support for every effort; in this respect the exercises which are employed in the education of deaf-mutes may be utilized. A general rule is that the learning of non-adapted but well-regulated movements is most useful for regulating adapted movements. Laisné had already made the

very correct observation that when the patients came from a great distance they profited much less by the gymnastics. This is because the education of the consciousness of movement demands attention, and because the attention, which is already deficient in choreics, becomes still weaker under the influence of fatigue liable to result from a long walk. It is therefore indispensable that the gymnastic exercises should never be carried to fatigue; the sessions must be short and repeated. Passive movements may prove very useful not only by their action upon the muscles but also because they recall regular motor images; after a movement has been passively executed several times its voluntary performance becomes not alone more rapid but more energetic," and this fact may be as readily demonstrated in hysterics as in choreics. Massage may be a useful adjuvant to the gymnastics.

Moderation of the gymnastic exercises is all the more imperative since the muscular weakness of choreics is well known, and since, on the other hand, systematic repose has proved favorable in the treatment of the disease and has been specially recommended by Seguin. Repose and methodical exercises do not at all exclude each other, for the gymnastics can be utilized only on condition that they are short in duration.

In grave cases precautions should be taken to protect the patients against injury, by keeping them at rest and sometimes even in beds with padded guards. When such forms appear during pregnancy it may at times be necessary to resort to the induction of premature labor or abortion and thus to cut short the chorea.

Congenital chorea and chronic chorea have thus far defied all therapeutic measures, but the patients suffering from these forms may still be benefited by careful hygiene.

TETANY.

The affection described by Dance in 1831 under the name of intermittent tetanus, then under that of essential contracture, or contracture of the extremities, is more generally known now by the name of tetany, given to it by Corvisart in 1852. The disease is constituted by a group of symptoms occurring under a rather variable form. Sometimes it is preceded by prodromes during a period of several months, or these may be present for a few days only.

Symptoms.

The early *prodromes* are arthralgic pains, a sensation of heaviness, formication, pricking sensations in the extremities, various sensorial

troubles recalling those of chorea and of hysteria, crises of amblyopia, and temporary deafness. The immediate prodromes consist of a sense of fatigue, general malaise, headache, and especially fibrillary contractions in the parts which are about to be attacked. Sometimes, especially during epidemics, the contracture appears without premonition; sometimes it first manifests itself on the occasion of voluntary movements.

Contracture.—Most frequently the contracture affects the upper extremities; the hand assumes a characteristic form which Trousseau compared to that of the accoucheur as it was about to be introduced into the vagina; sometimes, however, the flexion of the fingers is accentuated and the hand acquires the shape of that seen in hemiplegia with contracture, and the flexion may even be so pronounced that the nails make wounds in the palm of the hand (Hérard). It is very exceptional to find the hand fixed in extension. Sometimes the contracture is limited to one finger or the thumb. In slight cases the upper extremities are alone affected; but in the severe forms the lower extremities are also invaded, chiefly the feet and the toes which may be flexed or extended. The foot is usually extended in the equinus position, the toes being directed inwards; rarely the digital extremity of the foot is elevated. The thighs are exceptionally affected, being then fixed in forced adduction.

In the benign forms the contracture is limited to the extremities, but in the graver cases the muscles of the trunk may also be involved, those of the thorax as well as of the abdomen, and the diaphragm. We may observe in different cases opisthotonos, emprosthotonos, or pleurosthotonos; and dyspnœa is sometimes so extreme as to threaten suffocation.

It is only in rare instances that the contracture is limited to the lower extremities; but in a case of Béclard it was confined to the hip muscles of one side and simulated coxalgia. Mattei has also seen cases in which the contracture was limited to the abdominal muscles.

Sometimes the contractures are generalized and affect the muscles of the face, of the eye, of the jaw, of the neck, of the larynx, pharynx, and tongue. We may readily understand how in these cases the vital functions may be gravely menaced.

The intensity of the contracture is as variable as its extent. Often the will may triumph over it, but at other times it is impossible to impart any movement to the affected regions. The degree of contracture may also vary in the same subject; but in general it attacks both flexors and extensors, and the resistance is the same in one sense as in the other.

The contracture presents itself under the form of attacks passing from the extremities towards the trunk, separated by more or less complete intervals of calm; these intermittent periods are said to be more marked in adults than in children (Stewart). The attacks last for a period varying from a few minutes to several hours, and they follow each other in a series which may cease abruptly or gradually. In the case of an abrupt cessation the remission is more complete, and the loss of muscular power is less absolute during the interval. When the attack has ceased, even if it is the last one, there often remain a sense of fatigue, numbness, and headache. It is sometimes possible to provoke an attack by compression of the limbs along the track of the nerves and vessels (Trousseau's sign), a circumstance which suggests a comparison between the hyperexcitability of tetany and that of hysteria. However, it must be noted that several observers have found that contracture is provoked more easily, or even exclusively, by compression of the artery (Kussmaul). The electrical irritability of the nerve persists during the interval of the paroxysms.

The contracture is not always accompanied by troubles of sensibility, but most frequently there are cramp-like pains which are exaggerated by voluntary or passive movements. Furthermore there are frequently cramps in parts unaffected by the contracture. Sometimes there are pains along the course of the nerves even in the regions where no contractures exist. There may be formication, numbness, and pins-and-needles sensations, chiefly in the extremities, which are especially affected with anæsthesia; the diminution of tactile sensibility extends also commonly to the mucous surfaces. There may likewise be a diminution of thermal sensibility and analgesia. These sensory troubles, which often last weeks after the disappearance of the motor disturbance, are frequently most marked on one side.

In rare instances we note œdema and redness in the neighborhood of the articulations, especially on the dorsum of the foot which is in the equinus position. Sometimes there are profuse sweating of the extremities, a brownish discoloration of the face and hands, and falling of the nails. The bladder may in rare cases participate in the spasm (Trousseau).

The *tendon reflexes* may be diminished (Bernhardt), but they are as a rule normal; the cutaneous reflexes, on the contrary, are exaggerated. All the motor nerves which are accessible, with the exception of the facial, have their galvanic and faradic irritability exaggerated (Erb), especially at the time of the repetition of the crises and during their height. As the attack passes off the electrical irritability diminishes, and becomes normal when the spasm has ceased. We might attribute to a special irritability of the ulnar nerve the common

attitude of the patient during an attack, an attitude which recalls that provoked by Charcot by compression of the ulnar nerve in hysterical subjects with muscular hyperexcitability. The nerves of sensation and of special sense are also irritable during the attack (Schlesinger).

The *general symptoms* are, as a rule, limited to a persistent headache; but sometimes there is a rapid pulse which may be attributed to the pain. Fever, if present, is to be attributed to the causal trouble. In children the contracture may be complicated by convulsions.

Althaus has described, under the name of *tetanilla*, a modified form accompanied by intellectual changes, which is characterized by symmetrical clonic spasms, ceasing during sleep, by an exaggeration of muscular excitability and of the tendon reflexes, but without rigidity and without Trousseau's sign (spasm produced by compression of the artery or nerve leading to the part).

Prognosis.

The duration of tetany varies from a few days to several months; it is most commonly two or three weeks. The usual termination is in recovery, but there are often alternating periods of improvement and aggravation. A cure may take place suddenly following an attack of an eruptive fever. Death sometimes occurs when the spasms are complicated by convulsions. The etiological conditions are, however, the chief elements in the prognosis. Tetany occurring in consequence of rheumatism or of some debilitating condition, such as lactation or diarrhœa, is usually recovered from, but that which is consecutive to a thyroidectomy easily becomes chronic and fatal; that form which results from dilatation of the stomach is said to be always fatal (Stewart). We have remarked above that extension of the contracture to the muscles of the face and thorax increases the gravity of the affection.

Pathological Anatomy.

The anatomical lesions found at autopsy of individuals dead from tetany are very variable. Most frequently we find congestion of the spinal cord and of its envelopes, and sometimes of the entire cerebro-spinal axis. Weiss found changes in the anterior horns of the cord in the cervical region in three cases of tetany following the extirpation of a goitre. It is certain that the complex of motor and sensory troubles to the exclusion of cerebral symptoms marks a lesion of the cord rather than one of the brain. But we are confined to conjecture as regards the localization of the lesion.

Etiology.

The question of causation, whether we have to do with a lesion of toxic or one of infectious origin, is equally difficult to answer. Following the reports of Weiss and the results obtained by Schiff, Hoffmann, Horsley, and others, quite a number of cases of tetany have been attributed to an intoxication by retention of mucin resulting from disease of the thyroid gland. Total thyroidectomy has been shown to be invariably followed by tetany in the cat, while partial removal of this gland has never had the same result; tetany followed only when at least four-fifths of the gland had been removed (Eiselsberg).

Other neurologists, reasoning from the frequency of tetany as a sequel of epidemic diseases, have regarded infection as playing an important rôle.

An inquiry into other etiological factors may throw some light upon the nature of the lesion in this disease. Tetany has been regarded as a disease of childhood, but Trousseau found that it occurred with greatest frequency between the ages of seventeen and thirty years, and Corvisart met with it most often between seventeen and twenty-one years. Puberty seems to exercise a certain influence; the disease is rare after the age of forty years. It has not been determined whether tetany occurs more frequently in one sex than it does in the other.

In the cases reported by Feer, the affection appeared to be congenital. Heredity, direct and similar or indirect and dissimilar, has been invoked, especially by Bouchut.

Lactation is a common cause, but we cannot say whether it acts through albuminuria, through anæmia, or through fatigue. Menstruation and pregnancy are rare causes (Dakin).

Digestive troubles appear to have an undoubted effect in the causation of tetany. Tonnelé called attention to this relationship in 1832; and Trousseau attributed a special rôle to diarrhœa, which appears to act especially by the debility which it produces, although gastrointestinal irritation seems also to be of importance. Tetany may attack individuals affected with dilatation of the stomach (Bouveret and Devic) when there has been a too rapid evacuation of the organ by the tube (Dujardin-Beaumetz, Dreyfuss-Brissac, Martin), and may also occur in the course of peritoneal affections. If dentition be a cause of tetany it is probably so by reason of the gastrointestinal troubles which are common at this period. In cases of gastric tetany Bouveret and Devic have extracted from the stomach contents a substance, the inoculation of which in animals excites tetanic convulsions.

Several of the infectious diseases may cause tetany; among them are rheumatism (Steinheim, Trousseau), cholera, typhus, and intermittent fever. The spasm may even present itself under an intermittent form, justifying in such case the term intermittent tetanus given to it by Dance, who regarded the paroxysmal spasm as a sort of larval form of the malarial attack. The influence of cold has been remarked by a large number of observers.

An etiological factor which deserves special mention is contagion, which was observed in the prisons of Belgium in 1846, and in a girls' school at Gentilly in 1876. The latter epidemic was especially interesting because the school for girls and that for boys were near together, and yet the latter escaped entirely. These facts together with the presence of hysterical antecedents in a number of cases and with certain characteristic symptoms of this morbid complex would lend weight to the theory of the hysterical nature of the disease (Raymond). But while the nature of hysteria still remains under discussion, we may content ourselves with admitting that tetany and hysteria have many points in common, and often develop on the same soil.

Influenced by the researches of Soltmann, Escherich, Kassowitz, and Babinsky, some authors are inclined to regard tetany and certain spasmodic phenomena more commonly associated with it, especially spasm of the glottis, as manifestations of rachitis. In the statistics collected by Cassel the presence of rickets was noted in eighty-six per cent. of the cases of tetany; but concerning figures such as these, we may be justified in remarking that he who proves too much proves nothing.

Bad hygienic conditions in general play an important part in the etiology of tetany.

Diagnosis.

The diagnostic points of importance in tetany are the habitual absence of fever and the intermittent character of the attacks. These suffice to distinguish tetany from the contractures which accompany grave fevers or diseases of the cerebrospinal centres. Ergotism, which may occur in epidemic form among individuals nourished in common under poor hygienic conditions, presents a considerable analogy to tetany.

Treatment.

Our therapeutic measures should be addressed as far as possible to the etiological conditions. Whenever the cause can be discovered our endeavor must be to suppress it. Tonnelé, Gourbeyre, Riegel, and others have reported cases in which recovery followed the expul-

sion of worms from the intestinal canal. I have seen, as doubtless have many others, the disappearance of tetany after the removal of a carious tooth. A cure may follow the regulation of disordered menstrual function, the suppression of faults in alimentary hygiene, a rational treatment of diarrhœa or of dilatation of the stomach, and measures suitable for overcoming intoxications, whether from without or from within the organism. Isolation is urgently indicated in cases in which contagion seems to be an active cause. Treatment by phosphorus in cases coinciding with rachitis has seemed to give good results. Thyroid medication might be useful in cases in which tetany has followed thyroidectomy; in such cases also diuretics and purgatives are indicated.

The symptomatic treatment consists in the employment of both internal and external measures. The latter are liniments containing belladonna, opium, or chloroform, tepid baths, cold affusions, and the continuous current.

Internally we may give the bromides, belladonna, or chloral, by the mouth or in rectal injection (Baginsky). In very severe cases we may have to resort to inhalations of chloroform.

LOCALIZED SPASMS.

Apart from the convulsive affections which we have considered in the articles on hysteria and epilepsy, and in the earlier sections of this article, there are also tonic or clonic spasms which are limited to the area supplied by certain nerves, and which cannot be referred to any specific lesion, deserving therefore to be retained in the class of spasmodic neuroses. These spasms are generally not painful. We have already had occasion to refer to epileptic neuralgia of the face, or tic douloureux, in which pain is an important symptom; but by reason of its paroxysmal form as well as of its accompaniments, this spasm differs from those which we are about to study and in which pain, even if it exists, is but an accessory phenomenon.

Localized spasms may affect any of the regions of the body, the extremities as well as the face, but they are more commonly observed in the face and the neck.

Trismus.

Convulsions limited to the muscles innervated by the trigeminal constitute masticatory spasms, trismus, which may be tonic or clonic, unilateral or bilateral. The muscles affected, either together or separately, are the temporals, the masseters, and the pterygoids.

In tonic spasm, to which is applied more especially the name of

trismus, the teeth of the lower jaw are strongly pressed against those of the upper; the mouth is firmly closed and can be opened neither by an effort of the will nor even by force. In consequence of this alimentation becomes almost impossible, and we must take advantage of the interstices of the teeth to introduce liquid food. The tense masseter muscles form masses of ligneous hardness in the cheeks. Sometimes these strongly contracted muscles are the seat of a continuous pain. If all the muscles are not equally affected the lower maxilla may be drawn to one side or obliquely deviated.

In cases of clonic spasm the lower jaw is moved more or less rapidly and more or less energetically either in a vertical or in a horizontal direction, producing either a rattle or a grinding of the teeth. These movements are sometimes rhythmical, sometimes irregular in their production.

Masticatory spasm, either tonic or clonic, manifests itself in a large number of morbid conditions. Most frequently it is found associated with other spasmodic phenomena. It figures under both forms in general or partial epilepsy, and in the epileptoid stage of the hysterical attack. Under the clonic form it often forms one of the choreic manifestations; it is represented by the masticatory movements in incomplete epilepsy, and by the grinding of the teeth in meningitis, in general paralysis, and in the gouty.

The tonic form, or trismus, is much the more frequent, and is met with in meningitis, tetanus, tetany, etc. In a certain number of cases it forms part of the secondary contractures of hemiplegia, in which it occurs as a more or less pronounced difficulty in separating the jaws accompanied by lateral exaggeration of the masseter reflex. This contracture would be found to be less uncommon than is generally supposed if we would study with more care the movements of the maxilla in hemiplegic subjects.[12]

Isolated masticatory spasm occurs with greater frequency in the tonic form. Its cause is more commonly a local irritation, such as a wound or necrosis of the maxilla, disease of the temporomaxillary articulation, neuritis, compression of the nerves, dental caries, and especially faulty eruption of a wisdom tooth. More rarely it occurs as a consequence of superficial ulcers of the gums (Little). Sometimes the irritation is seated at a distance, as, for example, a wound of a finger, intestinal worms, etc. When a spasm has once been caused by one of these factors, cold appears to be capable of exciting it anew (Sarazin). Hysteria seems to act as a predisposing cause. In some cases permanent contraction of the jaw appears to be dependent upon syphilitic myositis. Semon has reported the case of a clergyman in whom spasm of the elevators of the jaw occurred only

during the act of speaking, the patient being able to speak only with the teeth closely pressed against each other. The presence of this sole exciting cause led to the assumption that the spasm was a professional neurosis.

All these tonic spasms are, as we have said, usually painless, or are at most accompanied by a distressing sensation of tension. I have, however, observed a case of tonic spasm of the masseters, occurring remittently, excited by active or passive movements of lowering of the jaw, which was accompanied by sharp pain.[13]

Clonic spasms present no diagnostic difficulties, but it is not the same with trismus which may be confounded with ankylosis of the temporomaxillary articulation; in such case, if we are unable to form an opinion from the history of the patient or from an examination of the muscles, we may resort to chloroform anæsthesia. The diagnosis of the cause is not so easy, but it is important since the prognosis rests upon it. These spasms are generally rebellious to treatment unless we can remove the cause, such as a carious tooth, intestinal worms, etc.

The therapeutic measures at our command are rather meagre. Improvement or even definite cure has resulted from counterirritation, the application of electricity in its various forms, and massage. Antispasmodic and narcotic remedies have usually proved unreliable. When trismus is very severe and of long duration we must employ mechanical means to separate the jaws in order to permit of food being taken.

Facial Spasms.

Spasms in the domain of the facial may be tonic or clonic. Unilateral contracture, which may occur with the secondary contractures of cerebral hemiplegia, with tetany, or with glossolabial hemispasm of the hysterical, is also rather frequent after facial paralysis *a frigore*. In this case the face little by little deviates in a direction opposite to that of the primary deviation due to the paralysis. The contractured side of the face is stiff and immobile, and the eye on this side is almost always closed.

In the case of clonic convulsions we must distinguish between partial and diffuse convulsions (Erb).

The diffuse convulsion is often designated by the term non-painful spasmodic tic (*tic non-douloureux*) of the face. It occurs often without any apparent cause. It may be hereditary (Piedagnel, Rosenthal) or familial (Blache, Delasiauve, Gintrac, *et al.*). Emotions may cause it, but it comes on most frequently under the influence of peripheral irritation, such as wounds and bruises of the face, especially

of the orbital region, compression by a new growth at the base of the skull, otitis, caries of the petrous portion of the temporal bone, or abscess of the parotid region. Any lesion affecting the facial or the trigeminal nerve may produce this spasm. Cold appears to be a rather frequent cause. Certain irritations of the cerebral cortex may be followed by spasm in the domain of the facial. I have seen an instance in which it followed upon a traumatism which had left a depression of the cranium in the region of the angular convolution, but had occasioned no other trouble.

Symptoms.—The characteristic symptom of painless spasmodic tic of the face is a rapid contraction of the muscles innervated by the facial. The forehead, the eyelids, the lips, the alæ nasi, and the cheeks are affected in varying degree by the spasm which is the more marked as it is unilateral. The contractions occur often in series interrupted by pauses of more or less complete calm. These series, which are of longer or shorter duration, and greater or less in number, constitute a species of attack which often appears spontaneously but may be provoked by voluntary movements, emotions, or fatigue. In violent paroxysms, and quite constantly after the affection has lasted for some time, muscles innervated by other nerves than the facial may take part in the convulsions, such as the muscles of the tongue, the masseter, and the sternocleidomastoid. On the other hand certain muscles supplied by the facial, such as the stylohyoid, the digastric, and the muscles of the soft palate, are frequently not affected.

As a general rule, in spite of the spasm, the voluntary movements may be executed in an apparently normal manner. But if we make an accurate dynamometric examination we find in fact that the force of these movements is diminished and that they are executed appreciably more slowly than those taking place on the sound side.

As a rule, the spasm is accompanied by no painful sensations. Occasionally a numbness or pricking sensation in the tongue is complained of at the time of the spasm. Erb has reported a case in which there were subjective noises in the ear at the time the spasm occurred. The spasm of the eyelids may be so strong as to give rise to subjective visual sensations caused by compression of the eyeball. In some cases the attacks occur during sleep as well as in the waking period; in other cases the spasms cease during sleep.

In a certain number of cases there are points of arrest, pressure upon which will cause the spasm to cease; as a rule, these points are the same as those found in cases of trifacial neuralgia. Graefe has also described other secondary points, developing in the course of the affection, which are more or less distant from the first and usually of less power.

The diffuse convulsion does not usually appear full fledged, but more often comes on gradually, having been preceded by local spasms. In a certain number of cases the spasm remains circumscribed, partial as it is called. The elevator muscles of the nostrils or of the lips, the zygomatics, or the orbicularis oris may be affected singly; and the same is true of the orbicularis palpebrarum. The spasms of the orbicular muscles present the peculiarity that they are, or at least appear to be on superficial inspection, bilateral, although they are connected with a well localized point of irritation. Blepharospasm is often preceded by a simple winking which gradually becomes accentuated.

The *course* of these convulsions is usually progressive and chronic; it varies according to the individual, and often presents alternating periods of calm and exacerbation. Frequently the convulsions last, with temporary remissions, throughout life. Even when they have apparently ceased we have always to fear a return. Sometimes the area involved in the spasmodic movements becomes narrowed, but the latter do not cease entirely.

The *diagnosis* of this condition is easy, but not so the discovery of its cause. Indeed, it is only when no cause can be discovered that facial tic deserves the name of neurosis. Of course, if any causal lesion can be found, it is to this that our therapeutic efforts must be directed.

Treatment.—Benedikt has made the assertion that all cases which have not become inveterate yield to electricity; but Erb, on the other hand, has observed cases resistant to all methods of electrical application. Electricity applied to the points of arrest is said to be of special utility in blepharospasm (de Graefe).

Duchenne of Boulogne has recommended intimidation as a means of arresting a commencing tic in children. De Graefe has reported a case in which a strong effort of the will was sufficient to arrest a blepharospasm.

Narcotic and antispasmodic remedies have often been tried without result.

Stretching of the facial nerve has been performed, sometimes with success (Eulenburg). Section or resection of the nerve has the disadvantage of leaving behind it a paralysis. Prolonged compression of the nerve has also been recommended.

Lingual Spasms.

Tonic or clonic spasms in the domain of the hypoglossal nerve have been observed. Tonic spasm of the tongue is encountered in cases of hemiplegia with contracture. In one case of paralysis

agitans I have noted a rigidity of the right half of the tongue. Spontaneous contracture of the tongue is not of rare occurrence in hysterical subjects.

Clonic spasms are more frequent; they are often associated with other spasmodic affections, but may be strictly isolated. Hitzig, and Charcot and Ball have reported cases of spasm of this kind dependent upon lesions of the cerebral cortex. Verneuil saw an instance of lingual and maxillary spasm consecutive to a depressed fracture of the skull. Clonic spasm of the tongue may be one of the convulsive manifestations of chorea, hysteria, or epilepsy.

Isolated clonic spasms are rather rare. Fleury and Vallin saw a patient in whom tonic and clonic spasms appeared whenever he tried to speak, or even when he formed an intention of doing so—a condition known under the name of aphthongia. There are a number of cases of lingual spasm recorded in medical literature by O. Berger, Erb, Remak, Mondorf, Seppili, Erlenmeyer, Ganghofner, Bernhardt, and others. I reported a case of this nature before the Société de Biologie in 1887.

The patient was a woman, 56 years of age, of nervous antecedents, who was seized with pain in the left masseteric region following extraction of a molar tooth on this side. The pain diminished, but there followed spasmodic movements of extreme depression of the lower jaw, especially marked on the left side. This depression occurred at intervals of longer or shorter duration, being sometimes so sudden and extreme as to cause dislocation of the jaw. At the same time with this lowering of the jaw there was a forward propulsion of the tongue which was rapidly extruded from the mouth and excessively elongated. These combined movements of depression of the jaw and extrusion of the tongue were rendered still more grotesque by the fact that the patient had a set of false teeth which was often violently thrown from the mouth when the spasm was intense. Gradually the intervals between the attacks became shorter, and no treatment was of any avail, the patient being obliged to wear an apparatus to keep the jaws closed, a small wooden tube being held between the teeth to afford passage for liquid nourishment. The spasm almost invariably returned as soon as this retaining apparatus was removed.

We know very little of the clinical history of these cases of clonic spasm, except that they appear to be quite rebellious to treatment. The prognosis of tonic lingual spasms, in which sometimes the tongue may be held immobile against the vault of the palate (Valleix), is no more favorable.

Spasm in the Domain of the Spinal Accessory Nerve.

Under the names of functional spasm of the neck, spasmodic torticollis, rotatory tic, clonic form of hyperkinesis of the accessory of Willis, there has been described a clonic spasm of the muscles of the neck, which may be limited to the sternocleidomastoid or to the supply of the external branch of the spinal accessory nerve, but which often extends to the muscles innervated by the cervical nerves. The name of functional spasm of the neck, which is the most vague, appears to me to be the most suitable, not only because of the absence of precise localization of the convulsion in most cases, but also because of its most frequent occurrence in connection with active movements.

Functional spasms of the neck are of rather rare occurrence, more so in women than in men. More commonly they appear between the ages of thirty and fifty years, but may come on either earlier or later. Sometimes they have been observed in children under an acute form and of rapid development, terminating in recovery (Smith). The nervous temperament, as revealed by a neuropathic heredity or by the personal antecedents, is a frequent predisposing condition; in a certain number of cases the affection acts like an hysterical symptom.

Etiology.—The determining causes are those which are capable of producing a deterioration of the general health, such as influenza or other infectious disease. The affection often coincides with neurasthenic troubles. In two cases the spasm presented itself under the form of periodical attacks occurring with intermittent fever, and subsided rapidly under the influence of the specific treatment of this condition. Cold and traumatism are often referred to as exciting causes. Other possible causes are lesion of the nervous centres, encephalic neoplasms, or lesions of the cervical portion of the spinal cord. Again, the spasm may be dependent upon a local irritation, rheumatoid pain in the muscles, or dental caries. Sometimes the irritation is more remotely situated, as in the uterus or in the intestine. The emotions may play a rôle in the production of these troubles, but their influence is seen most distinctly in the repetition and in the intensity of the attacks.

Symptoms.—The spasm is usually unilateral, or if the muscles on both sides are affected those on one side are more so than those on the other, and it is only in very rare instances that the two sides are equally affected. The muscles involved are generally the rotators of the head, but sometimes the posterior neck muscles are affected, giving rise to spasmodic retrocollis.

The sternocleidomastoid, which is the muscle most frequently in-

volved, causes rotation of the head, the chin being thrown forwards and upwards, and to the opposite side. Spasm of the upper portion of the trapezius turns the face to the opposite side, at the same time drawing the head backwards and slightly raising the shoulder and causing the scapula to approach the vertebral column. Affection of the splenius, of the levator anguli scapulæ, or of the posterior deep muscles rarely occasions any peculiar deviation of the head.

Quite frequently several muscles are affected at the same time. The most common combination is that of one sternocleidomastoid and of one trapezius on opposite sides; the rotation is then nearly horizontal; this rotation is very pronounced, the chin almost touching the shoulder, when one sternocleidomastoid is affected at the same time as the splenius of the opposite side. When the spasm involves the posterior muscles of the neck the face is directed upwards; in a case reported by Gowers this retroflexion was so marked that the face became almost horizontal. The levator anguli scapulæ and the scaleni may take part in the spasmodic combination, but they scarcely modify the attitude.

The spasm is often the only symptom present and has been preceded by no other trouble. Sometimes its appearance is preceded by pain, a feeling of weight in the nucha, or other troubles which are most commonly of a neurasthenic nature. Sometimes the spasm appears suddenly in full intensity, at other times it comes on gradually. The rapidity of the shocks varies considerably; they may be repeated every second or even more frequently. In one of my cases I counted seventy-two spasms to the minute. Sometimes these are repeated at regular intervals, sometimes in the form of more or less rhythmical series. The muscles may be felt as hard masses at the instant that the deviation of the head occurs.

Quite commonly the spasm is continuous as long as the patient is executing any movement whatever; at other times it is manifested in attacks separated by intervals in which the muscles are flaccid. In some patients there is absolute immobility in the intervals between the spasms; but that this result may occur it is necessary that the head, the trunk, and the upper extremities should be firmly supported. The least movement even of mastication or of articulation brings back the spasm, which is also provoked by strong impressions upon the organs of special sense, or by mental emotions; the reason of this is that every sensorial stimulation or mental representation necessitates muscular action. But specially active muscular movements are the common exciting causes of the spasm; thus a patient who is able to stand erect for some moments or even to take a few steps with his head immobile, is at once seized with spasms as soon as he com-

mences to walk upstairs. When once spasm is present it is seldom that a strong effort of the will can abolish it. Natural or artificial sleep is the only condition in which there is always a relaxation of the spasm. With a certain number of patients a slight support to the head even with one finger suffices to maintain the equilibrium; with others compression of the points of arrest produces the same result. I have seen one patient who was able to arrest spasm of the sternomastoid muscle by forcibly extending the head.

The extent of the movements varies as much as their frequency. Sometimes the oscillations are scarcely perceptible; at other times the rotation of the head is extreme, the chin being thrown around towards the shoulder, sometimes elevated, or sometimes depressed so as to touch the clavicle. In extreme cases of this kind symptoms of compression of the subclavicular vessels and nerves have been observed, as in a case reported by Romberg. In one of my patients the numbness of the arm was particularly distressing. Phenomena of this kind have also been attributed to spasm of the scaleni muscles.

The force of the spasm is also variable; sometimes it may be overcome by very slight resistance, at other times it is invincible. Sometimes the twisting of the neck is excessive, the skin of the nucha being thrown into deep folds.

Although as a rule the sternocleidomastoid and trapezius muscles are the ones affected, the other muscles of the neck being only exceptionally or accessorily involved, the spasm may be much more extensive. Not only may all the cervical muscles be affected but often the trouble passes beyond the domain of the spinal accessory. I have observed an extension to the domain of the hypoglossal nerve, to the supra- and subspinal muscles, to the rhomboid and to the muscles of the upper extremity. Gerhardt has observed spasms of the vocal cord on the same side as those of the external muscles. Not uncommonly there are various coincident spasms of the facial muscles.

As a rule the spasm is painless, but sometimes there are painful points more or less constant in the cervical vertebræ, or at certain points in the course of the spinal accessory or of the cervical nerves. Some patients complain of vertigo which might be referred to visual troubles caused by the rapid movements of the head. There have been observed in persons suffering from spasm of the neck, strabismus and ocular paralyses to which even a pathogenic rôle has been attributed. Some have permanent deformities such as facial hemiatrophy or scoliosis. Finally spasm of the neck may be found associated with various spasmodic affections of the degenerate, with the exclamatory tics.

Transitory aphonia has also been reported (Nègre), as well as a

difficulty in swallowing liquids. I have seen a case occurring with homonymous unilateral visual hallucinations which argued in favor of the cortical origin of the spasm.

As a rule functional spasm of the neck is a clonic convulsion, but it may appear under the tonic from (Debout). The patient may be able to hold himself still, but as soon as he begins to make movements the neck twists and keeps its vicious position. The two forms, tonic and clonic spasms, may occur in the same individual (clonicotonic torticollis of Benedikt).

Frequently the muscle which is most profoundly affected by the spasmodic movements appears to be increased in volume. In other cases we observe a diminution in volume of the opponent of the affected muscle. This atrophy of the side opposite the spasm may be the result of the relative inactivity of the muscle, but it may also play a causal rôle, as in paralytic torticollis. The etiological action of atrophy of the supposed healthy side may be supported by theoretical reasons as well as by clinical facts.

In unilateral paralysis provoked in hysterical subjects, there is often an increase of motor force on the opposite side, and in the clinical cases of hysterical hemiplegia we sometimes see that in proportion as the paralysis improves, the motor energy diminishes on the healthy side. This kind of balance of nervous action is not easy of explanation. I may recall the fact, however, that this observation is not unique; Claude Bernard noted an analogous fact in the course of his studies on the great sympathetic nerve: "But, at the same time as galvanization of the upper extremity of the sympathetic reduces the temperature of the ear on the same side, we see the temperature of the other ear rise." There is hardly need to recall the fact that stimulation of the sympathetic acts not only on the vascular supply but also on sensation and muscular tonicity.

The influence of weakness of the antagonists may also be explained by a mechanism analogous to that which presides over the common phenomenon of cramp. The spasm which we are now considering presents indeed a certain analogy with cramp—a painful contraction occurring also in connection with voluntary movements, but especially in connection with violent movements and under conditions which deserve a careful study." Cramps occur chiefly in the muscles of the calf, in the toe muscles, the plantaris minor, the quadratus lumborum, the muscles of the nucha, and in the genioglossus. Whatever may be its seat, when the cramp does not occur in the course of some violent movement, it takes place during a passive shortening of the muscle, as cramp of the geniohyoid during yawning, cramp of the calf muscles during coitus or in individuals who sleep curled up with the

joints of the legs flexed. Stretching of the muscle, which relieves the cramp or cuts it short, is an evidence of the effect which shortening of the muscle has in producing the condition. In short, cramp may be compared to paradoxical contraction. Westphal has described under this name a phenomenon which consists in contraction of the anterior tibial, causing adduction and a certain degree of dorsal flexion of the foot, occurring in certain individuals when a sharp movement of dorsal flexion is imparted to the foot; the foot remains in the position described for quite a long period. Westphal explains this phenomenon as produced by the sudden relaxation of the muscle affected, while Erlenmeyer attributes it to stretching of the antagonists; I need not dwell upon the arguments which may be adduced in favor of this latter opinion, but the fact remains that active or passive shortening of a muscle may provoke its contraction. That is what takes place in cramp, it is what takes place also in the case of atrophy or of congenital weakness of a muscle, although the effects of this latter may not be manifested until late and then under the influence of some accidental cause which accentuates the weakness. The same mechanism may be invoked in the case of the spasms which we are considering here, all the more as we may sometimes see an amelioration under the influence of faradization or of massage of the so-called healthy muscles. We may obtain a cure, often only temporary, after section or stretching of the nerves on the side of the spasm; but this operation is necessarily followed by paralysis of longer or shorter duration, and it is possibly in consequence of this paralysis that the restoration of the weak muscle, either temporary or definitive, may take place.

Apart from theoretical considerations, there are certain facts which demonstrate the reality of the rôle of impotence of the antagonists. I have observed a case of spasm of the neck occurring in an hysterical subject at the same time with a facial and brachial paralysis of the opposite side, recovery from the two conditions occurring simultaneously. In a woman affected with paralysis agitans of hemiplegic form in the beginning, there was a spasm of the neck which disappeared when the phenomena of muscular weakness extended to both sides. We do not pretend to make this explanation fit all cases of functional spasm of the neck; we only mean to show that alongside of primary spasms there may be secondary spasms the treatment of which presents special indications and should be directed to the paralyzed muscle.

The localization of the spasm in the muscles supplied by the external branch of the spinal accessory nerve has led to the designation hyperkinesis of the accessory of Willis (Jaccoud). Although the

spasm is not always thus strictly limited and all the muscles supplied by this nerve are not always involved, this pathogenic designation may be applied to a certain number of cases. We may readily suppose that, like the spinal accessory nerve, the other nerves in the domain of which spasm may be produced are susceptible to irritation at several points in their course.

The central origin of the spasm is rendered probable by a case reported by Poore[15] of spasmodic torticollis which persisted in spite of section of the sternocleidomastoid, because the splenius was also concerned in its production, which was cured by an antisyphilitic treatment. This torticollis coincided with headache of the frontal region on the opposite side, which disappeared at the same time as the spasm. We know that a certain number of cases of facial tic may be referred to irritating lesions of the cortex.

Brissaud[16] regards spasm of the rotatory muscles of the head as a mental torticollis, applying to it the general theory of Charcot: "The tic is a disease which is material only in appearance, it is in one sense a psychical malady." This theory, which has since been extended to hysteria, is well calculated to cause surprise, and it is not without interest to see upon what facts it rests. Bain has advanced the opinion that the idea of a movement is really the commencing movement. This proposition is capable of experimental demonstration.[17] We deduce from this that when we think of a movement the movement must take place; it is also legitimate to conclude from the necessary relation of movement to thought that we think only because we move. But admitting it to be as it is interpreted, the fact does not constitute all of psychology. Other facts are no more contestable than the preceding; such, for instance, as the thought is nothing else than a feeble sensation ("no sensation, no ideas," said La Mettrie); and there is no sensation without movement (no irritation, no movement, no ideas). The intervention of the idea which seems to precede the movement can in no way allow us to suppose the absence of an irritation, that is to say, of a material fact. In degenerates who have tics we may assume a lesion of evolution of the cortical centres causing their irritation or determining an irritable weakness of that region which renders it more sensible to stimuli of peripheral origin. But it remains to be proved whether degenerates are subject to an essential mental spasm from the mere fact of their degeneration, as Bompaire has claimed, although he has not noted any characteristic of degeneration, either physical or mental, in his patients. Furthermore, Bompaire admits in his conclusions that mental torticollis has no special clinical characteristics.[18]

In certain cases of rotatory spasm of the neck the movement may

be temporarily suspended by very slight resistance made with a single finger. But this spasm is not the only trouble which may be suspended by a slight peripheral irritation, and among these troubles there are some, like epilepsy, which have never been asserted to be purely imaginary affections. The mental theory of the etiology of nervous troubles of which we know no physical cause has the immense advantage of dispensing with all effort in the search for this cause, but it has the disadvantage of destroying all chance of finding it. It is a lazy theory with which we should content ourselves only provisionally. Since I have formulated these objections Brissaud and Meige[19] have published three new cases of mental torticollis, in two of which the torticollis was preceded by pains in the nucha, the third being preceded by a spasm of the arm which itself had been preceded by numbness. These facts are better adapted to combat the mental theory than to sustain it. Whatever causes pain may also provoke reflex spasm.

Isidor[20] ends his study on this subject by the following incoherent conclusion: "The hypothesis of a mental torticollis, advanced by M. Brissaud, seems to be very close to the truth in a large number of cases; but we believe that this theory, although it may explain the facts coming on during the course of the affection, is incapable of application to the origin, to the primary disease upon which have been engrafted the symptoms of a purely mental order." An argument in favor of the mental theory would be a collection of cases of cure by suggestion, but this collection has not yet been begun.

The symptomatic *diagnosis* of the affection is extremely easy. The functional spasm shows itself, indeed, as soon as the muscles of the neck enter into action; when suspension is produced by appropriate attitudes or by compression upon a point of arrest, the spasm is of short duration. The spasms of epilepsy are not of this character; they are not necessarily provoked by active movements, and they occur independently of these movements. The same is true of the convulsive tics.

It is more difficult to discover the origin of the spasm. We must determine whether we have to do with a primary or a secondary spasm. The atrophy of the antagonistic muscles does not constitute a sufficient sign, and acquires a value only when it is associated with other paralytic troubles. The atrophy or weakness may have its origin in a congenital defect or in an acquired paralysis, hysteria, paralysis agitans, etc.

Primary spasms may be the consequence of irritation of the cerebral cortex by a tumor or a syphilitic lesion, or in general paralysis, which lesions will betray themselves by other symptoms, such as

headache, vomiting, troubles of motility, of general and special sensibility, or of the intellect. When they are dependent upon a lesion of the cord, there are usually painful points and other phenomena associated with irritation of the nerves.

In the majority of cases we find no association which is of a nature to indicate definitely a localization of the cause. It is that which has caused the affection to be classed among the neuroses, and we are reduced to hypotheses as to its origin. From the fact that spasmodic torticollis is sometimes associated with other tics we are tempted to refer it to a cortical irritation, but this gives us no very precise indication for treatment.

Since the spasm is not due to an accessible cause, the *prognosis* is always grave. Cases in which a complete cure has been obtained are rare in spite of the most energetic treatment.

Treatment.—Medical treatment consists solely in the employment of remedies to quiet reflex irritability. Tonics, such as iron, arsenic and its derivatives, and antispasmodics, such as the bromides and belladonna, are among the remedies that may be tried. In order that any benefit may be derived from the employment of the last-named drugs they must be pushed to the limit of tolerance. Harley and Wharton Sinkler have obtained good results from conium given in the dose of sixty drops of the fluid extract in the twenty-four hours.

Revulsives have also often been employed. Busch has reported good results from cauterization of a large surface. Blisters applied along the vertebræ have also sometimes been of service.

Massage and faradization of the antagonistic muscles have often caused an improvement in the condition. Poore has also had good results from continuous currents applied to the spinal nerves.

The orthopedic measures consist in the application of retentive apparatus, with or without the intervention of tenotomy. Certain patients are made quite comfortable by an apparatus which gives them firm support. The effect of tenotomy is as a rule only temporary.

Surgical measures are directed specially to the external branch of the spinal accessory nerve, which may be ligated, stretched, divided, or resected. Of late years operations have been performed upon the posterior branches of the spinal nerves, following special indications furnished by the seat of the spasm.

Trepanation has been suggested for the relief of extreme cases; the centre for movements of rotation of the head is found anterior to that for movements of the arm, and is equally accessible.

neighborhood of the hip are excited by pain or local irritation in coxalgia or psoitis, and they are seen also in hysteria. Clonic spasms of these muscles are rare, although Klemperer has reported a case of convulsive tic of the psoas and iliacus muscles.

The *femoral triceps* may be the seat of tonic and also of clonic spasm (Erb, Eulenburg). Remak has observed gluteal cramp coinciding with other spasms on the same side. The flexors of the leg, the biceps, the semimembranosus, and the semitendinosus, may likewise be affected.

Contracture of the *peroneus longus* produces a peculiar form of talipes valgus with depression of the submetatarsal projection and elevation of the plantar arch, diminution of the transverse diameter of the foot on the line of the heads of the metatarsal bones, torsion of the foot with oblique folds on the plantar surface, deviation of the foot outwards at the articulation of the calcaneus with the astragalus, prominence of the tendon of the peroneus longus above the external malleolus, and secondary contracture of the peroneus brevis and of the extensor longus digitorum (Duchenne).

Tonic spasm of the muscles supplied by the peroneal nerve has been observed (Weir Mitchell, Erb).

Cramps of the calf (see page 697) affect the muscles supplied by the tibial nerve. The condition is one of painful tonic spasm, occurring, especially in certain states of the general system, under the influence of fatigue, but particularly favored by passive shortening of the muscles, which then become suddenly actively contracted, raising the heel and fixing the foot in a position of equinus with the toes flexed.

Spasm of the Respiratory Muscles.

Contracture of the *diaphragm*, occurring under the influence of cold or in the course of tetanus or rheumatism, is characterized by an enlargement, especially transverse, of the base of the thorax, by a projection in the epigastric and hypochondriac regions, and by symptoms of suffocation. The shoulders are raised, the head is thrown back, the cervical muscles capable of raising the upper part of the thorax are called into play, but without being able to prevent the gradual weakening and slowing of the respiratory movements, with finally asphyxia and death if the contracture persists. When diaphragmatic contracture is unilateral, the respiration is greatly impeded, but asphyxia is not to be feared. This contracture may yield to inhalations of chloroform or injections of morphine, or to application of the faradic or galvanic current, which should be made with the least possible delay.

Hiccough is caused by clonic spasm of the diaphragm, su sion of which occasions a noisy expulsion of air, followed complete inspiration and then by a normal expiration. to Erb, the condition is one of sudden and brief contractic diaphragm, producing an inspiratory noise suddenly interr closure of the glottis. The spasms are more or less rapid an They are often symptomatic of inflammatory lesions of the a viscera, or simply of gastric irritation. They are more co children and may occur in them through the influence of i

It is especially in cases of so-called essential hiccougl may see relief obtained by rapid and uninterrupted respirat ments (Mathieu), a spray of ether to the epigastric region (R other cutaneous revulsives, or by the swallowing of liquids ears are closed with the tips of the fingers.

Spasmodic convulsions may involve, at the same time diaphragm, all the respiratory muscles, even the accessory n respiration (Erb).

Sneezing is effected by a strong inspiration followed b and noisy buccal expiration. It is generally announced by tion of tickling in the nares. It is often repeated in a ser may be remarkably prolonged in hysterical subjects. It m among the other symptoms of the epileptic aura. It is usu flex phenomenon caused by irritation of the pituitary muco brane; but in a certain number of cases it is provoked by of more or less distant organs, such as the eyes, the stomacl testines, or the genital organs. Sneezing provoked by bri is regarded as a simple reflex; it is supposed that the cilia derived from the trigeminal, like the nerves of general sens the nasal mucous membrane, are the centripetal route of th but I have shown that this sternutation is more often the re double reflex action: first a lacrymal secretion caused by loc tion, and then sneezing provoked by the flow of tears into t cavity.[21] Peter Young observed attacks of sneezing occurr woman during two successive pregnancies and Romberg sa ilar case. The sneezing may be due to a lesion of the tri Thus Romberg reports a case of a woman who for four year ing a fall on the head had fits of sneezing; after death t found a lesion of the neurilemma of the third branch of nerve.

Yawning consists in a deep, sometimes noisy inspiration treme expansion of the thorax and elevation and throwing the shoulders, the inspiration being accompanied by a wide of the mouth. Sometimes at the same time the arms are ra

extended—pandiculation. This is an involuntary act which is often excited by imitation if it is seen or even heard in another, for we know that it is unconsciously imitated when heard by the blind as well as when seen by those who have their sight. Physiologically it is caused by fatigue, hunger, cold, excessive heat, or by depressing mental states. It is more frequent in women, especially when excited by imitation. Yawning is often a symptom of digestive troubles; it may also occur in a number of conditions in which hæmatosis is interfered with, at the beginning of asphyxia, at the moment of invasion of a fever, etc. It is frequent in psychoses of a depressing character, in hysteria, and in epilepsy. In certain cases it appears under the form of an attack in which it is not possible to refer it to any general condition.

Spasm of the respiratory muscles shows itself also under the form of cough, of laughter, sobbing, etc.; but these manifestations are always dependent upon some local lesion or a general state which is ordinarily easy to determine; in other words they are symptoms which can be referred to a special cause, and they therefore do not belong in the category of neuroses.

PROFESSIONAL NEUROSES.

In our brief review of the local spasms we have seen that certain of them, those of the neck, may declare themselves exclusively or at least in the majority of cases, under the influence of voluntary muscular movement. These functional spasms form a connecting link between ordinary spasm and the professional spasms which we shall now consider. We may also recall the fact that in certain cases of functional spasm of the neck we have had occasion to note the existence of paralysis of the antagonists; we shall find the same condition in the professional spasms, which have sometimes been called professional pareses because the paralysis sometimes takes precedence of the spasm. Sometimes also tremor is added. Writers' cramp is the type of these professional neuroses.

Voluntary movements may excite general spasms. Herpin reports the case of an engraver who was so often seized while working his burin that he had to abandon his occupation; the same thing may be caused by the act of writing, an example of which I reported to the Société de Biologie in 1891. But writers' cramp properly speaking has nothing to do with these general convulsions. It begins usually gradually; the fingers become less supple and more unmanageable; they become numb and stiff when the patient tries to hold the pen; then there supervenes a loss of power in which

sometimes spasm, sometimes tremor, and sometimes paraly
the most important part. At first transitory and caused by
longer or shorter duration, these troubles become graduall
nent, recurring immediately upon the patient taking up the

Writers' cramp occurs under the various forms of
tremulous, and spasmodic properly so-called. The paralyti
the least common. Duchenne knew a secretary who afte
written a few lines found it impossible to move the hand and
arm, although the fingers would still continue the writing
Another patient had a paralysis of the adductor pollicis occu
der the same conditions, forcing him to drop the pen. This
does not usually remain limited to the special function, but
extends. This paralytic form of writers' impotence may be
to the hephæstic hemiplegia of Frank Smith which occurs
prolonged use of a hammer striking rapidly repeated bl
which does not remain strictly localized.

The tremulous form of writers' impotence is characteri
sort of trembling or choreiform movements. In a case re
Duchenne, the patient, as soon as he began to write, had a t
of the hand and forearm which increased so long as the effor
was persisted in, and also when strangers were present. S
writing is easy for a considerable time, then fatigue sudden
itself, and disordered choreic or athetosic movements of th
are produced, the pen being jerked to a distance.

In the true spasmodic form there are discordant contra
different muscles: the index finger is extended or flexed
thumb executes movements in the opposite direction; the tw
nal phalanges of a finger are flexed while the first is extended
abnormal movements may extend to the entire hand, which
rapid movements of pronation and supination or is strongl
These movement are produced at first after the patient has b
ing for a long time, and are quieted by a suspension, writin
ing possible again after a short rest; but gradually it comes
promptly and finally appears as soon as the patient begins
or sometimes even the mental image of the movement, the
writing, suffices to provoke the spasm. Sometimes the sp
form follows the paralytic form, and we often note that the
nists of the muscles affected by spasm are themselves markedly

The spasmodic contraction is often indolent, it is also acco
in many cases by a very annoying sensation of weight, especia
the patient persists in writing. Sometimes there is a true
cramp, and some writers describe a neuralgic form, with lar
pains limited to the hand or extending along the course of t

and median nerves. In some cases there are spinal pains, chiefly in the cervical region; at other times there is a hemicrania of the opposite side. Permanent disorders of sensation, dysæsthesia or anæsthesia, are rare.

Writers' cramp is quite frequently associated with other spasms in other parts of the body, or with other motor or sensory troubles. Sometimes the fingers present the symptoms of local asphyxia or syncope; in cases of long standing there are sometimes permanent vasomotor troubles, manifested by passive congestions with a tendency to ulcerations, by dryness or hyperhydrosis or trophic troubles of the nails. Quite frequently we observe symptoms of a general neuropathic condition, the patients are often emotional, subject to alternations of excitement and depression, and suffer from insomnia, vertigo, or other neurasthenic troubles; in quite a number there are teratological stigmata of degeneration.

The information obtained by electrical examination is of a contradictory nature. Most commonly we find normal reactions, but occasionally there has been found the reaction of degeneration; at other times, however, there has been noted an increase of galvanic and faradic excitability.

Does the etiology of writers' cramp give us any information as to its nature? As its name indicates, it occurs in persons who write for a living. But it is erroneous to suppose that it affects only those who write very much. It has not been shown to be materially influenced by unhygienic surroundings of the patient. It occurs especially in those who are suffering from mental overwork and who show neurasthenic or neuropathic symptoms. Gallard has noted that the affection is more common among higher officials than among their subalterns who write much more than they. There is no question but that heredity or a congenital predisposition plays an important rôle in the production of this affection. Gallard has reported a case of a notary whose mother and sister were no longer able to write, being affected like himself. External conditions may awaken the predisposition; such are cold, traumatism, and compression of a nerve. I reported to the Société de Biologie, in 1887, a case of writers' cramp following repeated compression of the ulnar nerve in the forearm.

Writers' cramp has been localized in the central organs, not only because of the general neuropathic phenomena and of the systematization of the troubles, which, however, is not complete, but especially because of the rapid appearance of the cramp in the left hand when the patient has trained himself to write with it, and of the reproduction of the cramp in the right hand during the attempts to write with

the left. Nevertheless cases like that which I have reported, in which the cause was clearly peripheral, compression of a nerve, speak in favor of the peripheral theory of its production. Poore classes writers' cramp among the neuralgias.

Treatment.—Writers' cramp is very rebellious to treatment. Prolonged rest is still the best means which we have at our command. I have seen two cases cured by it; in one there was a cramp which appeared to be of peripheral origin, but in the other the affection was developed without any recognizable cause following violent emotion in a very nervous individual. I advise absolute rest, the hand to be carried in a sling; and when the spontaneous movements have ceased, or at the end of eight or ten days, if the spasms are not reproduced except during the act of writing, an attempt may be made to write; but this must be abandoned immediately if any spasmodic movement threatens. If the spasms are not reproduced these attempts to write are made at constantly decreasing intervals, but are always abandoned if the spasm threatens to return. This procedure is not often successful because it is usually impossible to obtain complete rest. Compression by an Esmarch bandage (Dalby) may be useful in sustaining the patience of the individual. In any case it is well to come as near rest as possible by having the patient use an apparatus which will enable him to hold the pen easily and which will necessitate very little tension of the muscles. There are various forms of apparatus, the choice of which must depend upon individual experience. Large and light penholders are to be recommended, but it is often necessary to hold the penholder in a new position, between the medius and index fingers, for example, in order to avoid production of the spasm.

It goes without saying that many cases of cure have been attributed to electricity employed in various ways, such as prolonged galvanization of the muscles and of the nerves supposed to be affected, weak currents through the spinal cord, the nerves, and the muscles, faradization of the muscles, central galvanization, etc. Static electricity may be useful in the same way as hydrotherapy, as a tonic.

Bouchut has claimed that many persons have been cured by means of a penholder of copper, or of copper surrounded by a band of zinc, or by one of gold or silver. These cases recall the metallotherapy of Burq.

Strohmeyer is said to have obtained success by means of tenotomy; but Dieffenbach, Langenbeck, and others have had only failures to report.

Narcotics and antispasmodics have been found to be of no service. Revulsives such as punctate cauterization of the affected region ap-

pear to have been useful, possibly by reason of the enforced rest following their application.

I have deferred mention of massage because I wish to speak of it in connection with the method of Wolff, which enjoyed a moment of notoriety and then fell into oblivion because it did not cure all the cases; but it is nevertheless often of great utility. It consists in an exercise at once active and passive, and in massage. Romain Vigouroux has described a method in a report of two cases successfully treated in this way.[22] 1. Three times a day the patient should execute a series of movements of the upper extremity successively in all directions, the hands being sometimes open, sometimes closed. The number of movements of each series, consequently the duration of the séances, is progressively increased and varies according to the individual case. The passive movements consist in more or less forcible stretching of the affected muscles; this stretching should be moderate but repeated very frequently by the patient himself. 2. The massage consists chiefly in friction and in tapping of the muscles. The writing exercises begin as soon as the spasmodic condition is notably diminished, that is to say, very early in the course of the treatment.

As is readily understood, most professional cramps affect the hand, and in the greater number of them we find spasmodic and paralytic phenomena more or less directly connected. I explained a professional cramp which I observed in a flute player as the consequence of a paralysis from exhaustion, and a number of other cases are capable of the same interpretation. Among musicians, such as pianists, violinists, and flutists, it is especially the muscles of the fingers which are affected, and the same is true of typewriters, telegraphers, clockmakers, drummers, embroiderers, cigarmakers, milkmaids; Poore, however, cites the case of a tailor in whom the subscapular muscle was affected, twisting the arm violently inwards. In gymnasium instructors and fencing masters it is the muscles of the forearm which are especially affected. In ballet dancers the cramp attacks the muscles supplied by the tibial nerve, especially the flexor of the great toe. In the case of a turner the flexors of the foot contracted as soon as he placed his foot on the pedal to turn the lathe. But it is not the muscles of the extremities only that may be affected with professional spasm. Seeligmüller has reported the case of a young girl who had spasms of most of the muscles of the neck and face after she had used them several days in making grimaces. In cases of this sort it seems to be a kind of obsession by the motor images. Spasm of the upper eyelids has been noted in a person who had worked long under artificial light; and in a person exposed to like conditions there were diplopia with contracture of the internal rectus, movements of the

eyelids, etc. Professional spasms of the tongue and larynx have also been observed. As Weir Mitchell has observed, voluntary movements may give rise to spasms of the most varied order.

CONVULSIVE TICS.

Until Charcot's time there had been described hardly any but facial tics and a few isolated tics of the extremities. It was under his inspiration that Gilles de la Tourette and Guinon wrote an account of this affection which is characterized by an involuntary but conscious repetition, by crises interrupted by variable intervals of sudden incoördinate and arrhythmical contractions of the muscles, more or less numerous, of the face, the trunk, and extremities, or of the vocal organs, often reproducing reflex or habitual movements. These spasms coincide in general with an impulsive tendency to imitate gestures (echokinesis), words (echolalia), or to ejaculate obscene words (coprolalia), and with a peculiar mental state in which attacks of obsession hold an important place.

Symptoms.

The tics predominate in the head which is jerked in various directions by spasms of the neck muscles, suddenly twisted or flexed on the chest by the contraction of one or both sternocleidomastoid muscles, thrown backwards by spasms of the nuchal muscles, etc. The face is almost always affected by sudden jerky spasms shown by the wrinkling of the forehead, frowning, closing or excessive opening of the eyes, rolling of the eyes, grimaces and various movements of the lips accompanied by a jerky laugh or a whistling sound, alternate movement of opening and closure of the mouth, movements of mastication, grinding of the teeth, projection of the tongue, and guttural spasms with snorting, sniffling, etc. The upper extremities are less frequently affected than the head. The shoulders may have, singly, simultaneously, or alternately, movements of elevation, of propulsion or retropulsion, and twisting as if the patient were trying to scratch himself by rubbing the clothes against the skin. The arms may be raised or moved in various directions, but always with the same jerky suddenness; the hands make scratching motions or gestures as of contempt or defiance, and the fingers rub noisily against each other. The lower extremities are more frequently exempt, although many patients make queer movements, rest themselves suddenly on the hip, strike the ground with the foot, extend the leg violently as if kicking, jump, take a few dancing steps, or stoop suddenly. In some cases the gait is extraordinary, the trunk oscillates from side to side in a

jerky manner, while the lower extremity of the opposite side kicks out, or walking is interrupted from time to time by a jump with both feet together or by a hop on one foot. In general, in the same attack several groups of muscles are affected simultaneously or successively; when the movements are made in succession they are often in the same order in each attack. In a certain number of cases the movements are limited to one side of the body, when they occur in the extremities, but this is rarely the case when they take place in the face. It is very exceptional to find the spasms generalized (Kahler).

Notwithstanding the impetuosity of the movements and their impulsive character, it often happens that the patients are able to execute correctly movements of the most delicate character; for example, the handwriting may not be altered in the least, one patient may be able to play the piano, another to use a graver's tool; Guinon mentions one who could juggle with knives without making any slip.

These involuntary movements are not unconscious, for the patient is perfectly aware that he is making ridiculous gestures, and he is sometimes even able to suspend them for a time through a violent effort of the will; but this effort is extremely fatiguing and is accompanied by a feeling of distress and malaise which is almost intolerable. The patients can usually more easily avoid the tic by making some voluntary movement than by keeping still. As far as possible also they endeavor to mask the tics by certain voluntary movements, but most commonly they are reduced to the invention of more or less plausible excuses to escape the ridicule of their companions, although the suddenness and the meaninglessness of their movements prevents their explanations from gaining credence. It is then that the patient seeks solitude in order to hide his infirmity. This is a useful measure also, as solitude tends to quiet the spasms which are, on the other hand, increased by excitement, surprise, or emotion of any sort; often the mere questioning of the physician suffices to excite repeated paroxysms. Certain somatic conditions, such as the menstrual period or an enfeebled digestion, may also provoke the tics, which, on the contrary, often cease during the course of any of the febrile affections. We may say that the tics always cease during sleep; nevertheless I know of a case in which there are sometimes attacks during sleep which seem to be caused by dreams.

Tics of the face and of the extremities are usually the first phenomena of the disease, and sometimes they remain the only ones; but frequently they are accompanied by disturbances of speech. The entire malady may consist in an exclamatory tic, which may remain for a long time as the only symptom; for example, I had under observation a man who for years would explosively articulate the name

of a young girl for whom he had had a precocious passio
dinarily the exclamatory tic coincides with other tics, and
in inarticulate cries, imitations of the noises of animals, inc
words, or scraps of incoherent phrases referring to nothing in
ular. It is the same with the involuntary exclamations as
tics of the members, the patient may arrest them for a mom
avoid them by the voluntary movements necessitated by conve
but his power of resistance is usually very limited, and in
time the conversation, like the muscular movements of his o
life, is interrupted by an inopportune spasm, and incoherent
or cries may make an explosion in the midst of the conversatio
he is sustaining. Quite commonly these verbal explosions co
obscene words (coprolalia), which are some times repeated o
another in a series, and are often ejaculated at most inopportu
ments. At other times the verbal explosion is the consequen
spasmodic idea; the patient, for example, may receive a visit
spasmodic expression which is uncomplimentary or worse, and
he earnestly desired to suppress.

A certain number of patients are driven in spite of themse
repeat, like an echo, noises which they hear (echolalia), whet
cries of animals, the sounds of nature, or spoken words. They
usually, the last words of the phrases pronounced before t
which they have spoken themselves. Sometimes the repetitio
a certain number of consecutive words at the end of the phrase,
times it is the last word only which is repeated one or more
or again, it is a certain word in the sentence which has impress
patient with special vividness; in a case of this kind which
observed the patient repeated only the proper names which
heard for the first time, and in another case only numerals we
peated.

Imitation in the subjects of tics is not exclusively of soun
some have an uncontrollable impulse to reproduce gestures or
ments which they see executed (echokinesis). I have observed
tient who asserts that most of the gestures which he executes ar
the realization of visual representations of imaginary scenes
those long past, while he rarely repeats movements which h
made in front of him.

These phenomena of imitation and of repetition of gestures ar
in the affections described in America under the name of jum
malady, in Malay under the name of latah, in Siberia under the
of myriachit, all of which appear to be of the same nature
malady under consideration.

Such persistent and intense motor disturbances can scarcely

the intellect intact. The fixity of attitude is indispensable to the exercise of attention of the will, which is diminished in the subjects of tics. All continuous intellectual application is impossible to them, many even become unable to read. Their conversation is often strikingly incoherent. Memory, association, and judgment are affected in the same degree as attention. But their mental state is also troubled by spasmodic ideas which present themselves periodically in the same way as their bizarre attitudes and which they are equally unable to restrain by their own efforts. These psychical tics may influence the conduct and lead to strange, ridiculous, or even injurious impulsive acts. They manifest themselves under the form of obsessions, of phobias, or of impulsions. In common with the motor tics they are irresistible and also conscious. Most common among the psychical tics are onomotomania or obsession of names, arithmomania or obsession of figures which may be manifested under different forms (Charcot and Magnan); anxious searching for a word, or obsession by a word which imposes itself constantly upon the patient, who is forced to ejaculate it suddenly; terror of a word which the subject seeks to avoid pronouncing, or belief in the preservative power of a word; and the indigestion of a word which is spewed out with loathing whenever it recurs. Among the obsessions is very frequently the search for the wherefore. Among the most frequently observed phobias are agoraphobia or fear of open spaces, and mysophobia or fear of soiled objects. The automatic and impulsive acts are of the most varied sort. The obsessions and automatic acts have often a physical basis; phaneromania, for example, is an obsession which is often provoked by projections from the surface of the skin or of the mucous membrane, or by anomalies of development; it is shown by an incessant and aimless desire to scratch some fleshy or corneous projection, to pull out a hangnail or to bite the nails, which are moreover usually too thin or defective. The remembrance of a reprehensible act may provoke an obsession just as may a physical deformity.

In its evolution the *maladie des tics* presents numerous varieties. More commonly there are only convulsive tics; sometimes spasmodic exclamations exist alone for a long time; and when the symptom complex is fully developed it usually comes on in a fairly regular order, first tics, then exclamations, coprolalia, echolalia, echokinesis, and fixed ideas. The evolution of the malady occurs sometimes in a continuous manner, sometimes by fits and starts, interrupted by intervals of improvement or even of perfect calm. In general, however, the affection grows steadily worse. While, in a certain number of cases the various symptoms may remain isolated, it may happen that others are added to them; I have observed two patients in whom

there arose visual hallucinations which were remarkable by the suddenness of their appearance and disappearance; in another patient attacks of epilepsy supervened. In a certain number of cases the fixed ideas end in actual insanity.

Prognosis.

The tic malady is rarely cured and it is very subject to relapses. When it is limited to motor or verbal troubles it may be compatible with a long life; a case of coprolalia is reported in which the patient reached the age of ninety. But when there are disturbances of ideation the disease may end in insanity. Obsession and impulse are conditions of irresistibility varying in intensity, which characterize the malady. When a struggle is made it often happens that the affection becomes only the more distressing; the patient wears himself out in a contest in which he is finally vanquished, and his resistance to the tic throws him into such a state of anguish that he is sometimes impelled to thoughts of suicide. In order to avoid this struggle most patients seek solitude; when they are alone they experience a great feeling of relief and may yield to their impulses without restraint.

Etiology.

The affection occurs rather more frequently in the male sex. It begins usually between the ages of five and fifteen years. It may be regarded as a malady of evolution, a result of congenital malformations whether hereditary or caused by conditions of malnutrition in the parents. Sometimes the malady may be referred in its origin to diseases occurring in infancy. Quite commonly the developmental predisposition is shown by somatic stigmata or teratological malformations. Certain accidental causes may provoke an outbreak of the trouble; such are physical or moral shocks, infections, intoxications, or imitations, but these play only a subordinate rôle.

Diagnosis.

The malady should not be confounded with localized spasms which we have studied above, which are uniform in their character without mimicry, limited to one muscle or to a group of muscles supplied by the same nerve or by nerves of neighboring origin which are capable of being affected by the same irritative lesion. It differs also from the coördinate tics, which are much slower and which consist of vicious habits, often provoked by a local irritation which may have disappeared later; nevertheless, we must recognize that the coördinated tics often closely resemble automatic acts associated with obsessions,

and are nearly related to psychical tics, although they may be repressed by exercise of the will.

In paramyoclonus multiplex (see p. 729) the face is rarely affected, the muscles involved being usually those of the lower extremities. It consists of muscular shocks which have no relation to the ordinary movements, and which are often themselves determined by movements of the limbs. Spasmodic exclamations and mental troubles are wanting.

Sydenham's chorea and chronic chorea, which have already been described in the early part of this article, differ entirely in the character of their movements from the malady under consideration.

Hysteria may resemble this affection in many of its manifestations. Hysterical rhythmical chorea has crises of movements reproducing habitual actions, malleatory chorea, saltatory chorea, etc., but its regularly rhythmical movements differ from the sudden and arrhythmical shocks of the tic malady. There is, however, a true hysterical conclusive tic, which is curable, the nature of which may be recognized by the presence of stigmata; but we must not forget that hysteria may coexist with the tic malady, and that the curability of the latter can only be assumed by the parallelism of the variations of its course with those of the progress of the hysterical manifestations.

Treatment.

Convulsive tics are usually incurable. The most favorable conditions for their improvement are isolation and change of surroundings, physical and moral discipline, application in some mechanical way or the study of some favorite subject if the patient is sufficiently intelligent. Hydrotherapy is an excellent agent of physical discipline; regularity in the habits is particularly to be recommended; gymnastics and passive movements may also be of service. A tonic, non-stimulant regimen is indispensable. The effects of medical treatment are almost nil. Bromides, chloral, opiates, and the like may quiet the attacks, for any remedy capable of suspending the functions of the nervous system will suppress the manifestations of this affection, but their action is only momentary, and is nothing of the character of a permanent cure. I have seen several cases of alleged cure by hypnotic methods which would not excite the envy of the most incurable of the subjects of the maladie des tics.

PARALYSIS AGITANS.

This affection, which was described by Parkinson, is characterized by a peculiar form of tremor and by permanent disturbances of motor activity.

At the autopsy of persons dying in the course of paralysis agitans lesions of the most varied sort have been found: scleroses, localized or diffuse atrophies of the spinal cord; lesions of the ependyma with obliteration of the central canal; sclerosis or softening of the pons; cerebral atrophy; or lesions of the same nature in both the cord and the encephalon. Recently Dana found a diffuse, chronic, and progressive interstitial myelitis, supposed to be due to microbic toxins or to the products of malnutrition. Sass found a focus of softening in the bulb from arteriosclerosis, at the same time with a sclerosis of the white substance of the cord and an obliteration of the canal of the ependyma, a chronic interstitial peripheral neuritis, and a more intense myositis than is generally found in the aged; and this led him to a belief in the possibility of a peripheral origin of the affection, a supposition which was strengthened by the rather frequent occurrence of neuralgic pains at the beginning of the malady. This great variety in the lesions together with the complete absence in many cases of any appreciable lesion, is a sufficient excuse for the retention of paralysis agitans in the indefinite and provisional group of the neuroses.

Etiology.

The etiology of paralysis agitans is as vague as its pathological anatomy. It is unquestionably more frequent in those who have passed the age of sixty years and very rarely occurs before the age of forty, but it may appear in youth, and Huchard has reported a case in a child of three years. Cases seen in adolescents are tolerably numerous. Sex appears to exert no special influence; it is generally believed that the disease is more frequent in men than in women, but there is no statistical support for this opinion. It is also uncertain whether it is of more common occurrence in northern climates.

All the conditions capable of causing rapid deterioration of nervous force may figure in the etiology. Moral shocks appear to have a specially injurious influence. Not only violent emotions caused by events of war or of civil strife, but also prolonged domestic discord may cause it; the action of sudden shocks is, however, more manifest, for frequently the tremor follows them immediately. Irritation of the peripheral nerves or traumatisms may play a similar rôle, and

their influence is often made manifest by an initial localization of the tremor in the wounded region.

Cold, whether acting suddenly and intensely or slowly and for a long time, has an undoubted effect in producing the disease (Betz, Charcot).

General affections may also have an influence upon the origin of paralysis agitans; I have seen the disease follow an attack of influenza in a woman thirty-three years old, and follow an acute tonsillitis in a man of forty-six years. Lannois saw the disease develop in a child of twelve years after measles. In a woman, forty-two years old, the tremor first appeared after poisoning by the fumes from a charcoal stove. A man, sixty-four years old, who previously had had only some slight oscillations in the left thumb, observed a typical tremor appear in the two hands after a brief attack of seasickness.

The etiology of paralysis agitans is, however, dominated by a morbid heredity. It is rare to observe direct and similar heredity, but paralysis agitans is met with in families in which organic diseases of the nervous system, neuroses, and diatheses prevail. The combination of rheumatism and paralysis agitans is rather common, and the treatment of the rheumatic diathesis appears to influence happily the progress of the neurosis (Pierret, Vesseli). The relationship of paralysis agitans to the neuropathic family is shown not only by coincidences in the family, but also by coincidences in the individual; thus cases have been reported of the coincidences of tabes (Heimann, Placzek) and of hysteria (Chabbert); I know of the case of a patient with paralysis agitans who, at the age of fifty-nine years, four years after the beginning of the tremor, commenced to suffer from epileptic symptoms in the form of ambulatory vertigo and convulsive attacks.

Symptoms.

The initial phenomenon of paralysis agitans is tremor. It is only exceptionally that this is accompanied or preceded by peripheral rheumatoid pains. The onset may be sudden or gradual, general or local. The sudden onset is seen generally following a violent physical or psychical shock; the tremor affects either all the members at once or a more or less limited region, most commonly then one thumb. Most frequently the onset is gradual, and the tremor is then generally localized. Sometimes it is remittent, being interrupted during a longer or shorter period, to reappear on the occasion of some emotion or shock or without any appreciable cause. Little by little the tremor extends, sometimes under the hemiplegic form, sometimes under the paraplegic form, but rarely under the crossed form (Charcot). At

the same time that the tremor extends it installs itself in a pe manner. It is only exceptionally that trembling involves the

The tremor is often accompanied from the beginning by a ing of motor activity and by a sensation of fatigue. This s of fatigue, accompanied by real weakness and sometimes by less severe rheumatic or neuralgic pains in the members t affected, sometimes precedes the appearance of the tremor. cases we may question whether the fatigue and the pain belong among the symptoms of Parkinson's disease, or whet are not the consequence of the depressing conditions which so often over the development of this affection.

Whatever may be its mode of onset, the tremor remains o most important symptoms, and its characters, most clearly d by Charcot, are of the greatest service in establishing the di It is manifested during repose and is suspended during the e of voluntary movements. As time goes on voluntary moveme be troubled by the tremor, but they do not increase it. E the tremor has become continuous during the waking hours during sleep. The tremor is usually increased when the attention is directed to it either spontaneously or on the occ a medical examination. Some patients are subject to ac oxysms of trembling excited by various influences or occurri out apparent cause.

The tremor consists in oscillations of slight extent, rhythm regular, and almost uniform in frequency in all the member are affected. It is a slow tremor of four or five oscillations per The attitude and movements of the hand are peculiarly charac The tremor of paralysis agitans is synchronous in all part body and follows the measure beaten by the foot. The phal the four fingers are in extension while the fingers are flexed a a right angle on the metacarpus; the thumb is carried towards of the palm, and the hand as a whole assumes the attitude th when holding a pen. The oscillations of the tremor give to t an appearance recalling that which it assumes in certain c acts, such as rolling a pencil between the fingers, twisting a t worsted, or crumbling bread.

It is generally admitted that the tremor is suspended by vo movements at least during the early stages of the affection; n less from the very beginning of the affection, although the appears to be normal, if we examine it with a lens we shall see characteristic irregularities; although the direction of the strokes is regular, we see that the line forms slight indentat that it is alternately heavy and light. Later the sinuosities i

and become readily visible to the naked eye, even while extensive voluntary movements still seem able to arrest the tremor.

In reality voluntary movements act more upon the amplitude of the tremor than upon its existence. I have recorded the tremor in a patient in whom voluntary movements appeared to have a suspensive action by having him push with his index finger a needle on a dial placed vertically before him. The record showed that the oscillations diminished considerably in extent during the performance of the voluntary act, but that they did not disappear completely. The character of the writing would suggest that the peculiarity is general in all the affected muscles.

The freedom of the head from involvement is less absolute than was formerly supposed. Charcot regarded the immobility of the head as a pathognomonic characteristic, explaining its occasional agitation by transmission of the tremor from the trunk; it is unquestionable, however, that the head may have a tremor of its own. Westphal, Villemin, and Demange have reported cases of the sort, and I have observed an undoubted instance. The facial muscles, as well as those of the jaw and of the eyelids, may be affected. The tongue may also be involved, but it trembles more when protruded than when it lies in the mouth. This fact shows that the law of suspension during voluntary movement is far from being absolute. The fact remains, however, that visible movements of the face and head are exceptional. The head is ordinarily immobile and as if soldered to the body. The movements of rotation are usually difficult and slow; the face is generally inclined forwards and a marked resistance is felt when it is attempted passively to extend the neck or to rotate it.

The Parkinsonian mask is characterized by an expression of hebetude and of sadness, sometimes of a majestic and dignified tranquillity. The look is fixed, the lower jaw is pendent, the cheeks are flaccid; in many cases the saliva dribbles incessantly from the mouth.

Speech is slow and staccato, sometimes explosive or tremulous and interrupted. There is no question that transmitted movements may play an important part in these speech defects, but the mobility of the lips and tongue may be affected, not only by the tremor but also by rigidity. Furthermore, the mental condition may not be intact.

Deglutition seems to be unaffected for a long period, but when the disease is advanced the patients often choke, and there is a resulting difficulty of alimentation which plays an important part in the production of the progressive loss of strength.

At a late period in the course of the disease we often find a difficulty of respiration which appears to be due to the weakened state of the thoracic muscles. In two patients whom I have observed recently

I noted that the excursions of the diaphragm were limited in what were apparently very deep respirations, but which were in reality very shallow ones (Litten's diaphragm phenomenon).

It is not only the head which is bent and stiffened, but the torso is also inclined forwards, and its movements of rotation are very limited, the patient turning the body as one piece by moving the feet. The upper extremities have also a fixed attitude; the elbows are slightly separated from the body, the forearms are flexed at an obtuse angle, and the hands, flexed upon the forearms, rest against the anterior part of the body. The thighs and legs are also in an attitude of more or less pronounced flexion. In some patients the flexion of the trunk is such that it makes a right angle with the thighs. Sometimes the patients hold their hands clasped behind them.

Alongside of the attitude of flexion, there are sometimes others. Writers have described a type of simple extension (Charcot and Richer), a type of extension with throwing of the head backwards (Dutil), a type of extension of the trunk and members with flexion of the neck and head (Bidon). These different attitudes may also change from one to the other. They are due, as Charcot remarked, to rigidity of the muscles. The rigidity, which appears soon after the tremor has become rather extensive, is often announced by cramps followed by stiffness. These phenomena, which are at first temporary, become later permanent; they may occur in true paroxysms which are sometimes very painful. As time goes on this rigidity may cause complex deformities; the hands may acquire certain deviations and deformities which resemble those of chronic rheumatism so closely as to be mistaken for them. The different segments of the lower extremity are often fixed in adduction and semiflexion with equinovarus deviation of the foot and hammer-toes from extension of the metatarsophalangeal and flexion of the interphalangeal articulations. The fixation of the legs in adduction may, especially in women, give rise to very great inconvenience.

This rigidity of the muscles, which plays the chief part in the production of the Parkinsonian mask, is sometimes especially marked in the occipitofrontal region, giving rise to the formation of deep transverse wrinkles of the forehead (Richer). It also sometimes involves the lingual muscles. I have seen a case in which this rigidity of the tongue was limited to one side and resulted in a very marked impediment to articulation.

The rigidity of the facial muscles is responsible for the strange hebetudinous expression, or that occasionally seen of terror or anger. In general the physiognomy of the sufferers from paralysis agitans is bad-tempered and disagreeable, in keeping often with their dispo-

sition, which is soured by the long continuance of their incurable infirmity. They are also usually capricious and self-willed.

This muscular rigidity, which imposes on sufferers from Parkinson's disease certain attitudes which we have just been passing in review, also prevents them assuming some of the habitual positions of the well. They can never lean on their elbows nor cross their legs. The stiffness and the fixed attitudes of paralysis agitans may exist without tremor, but we never see tremor without rigidity (Charcot). P. Richer[23] has made a detailed study of the attitude in paralysis agitans from which we may quote what follows:

"All the muscles seem to be contracted, and muscular relaxation is nowhere apparent. Everywhere, in the extremities as well as in the trunk, in the synergic or antagonistic groups, the muscles appear stiff, showing the bundles of which they are composed, and separated from the neighboring muscles by depressions which give to the body somewhat the appearance of being flayed. We may thus see the pectoralis major, the gluteus maximus, or the deltoid standing out with all the various bundles of which it is composed distinctly marked, as though they were separate muscles. The supinator longus forms a characteristic projection, the cord which it forms in the arm being almost a pathognomonic sign. In the arm the biceps is contracted in spite of pronation of the forearm, a paradoxical fact since normally the biceps produces flexion of the forearm only when its supinating action is not interfered with, that is to say, when the forearm is already in supination. Posteriorly the triceps is as rigid as the biceps, and so all over the body. The lower extremities furnish a no less interesting subject of study, all their muscles standing out prominently. The position of the knees is a simple one resulting from contraction of the quadriceps femoris. On the dorsum of the foot the tendons of the toes stand out, while the toes themselves are curved in towards the ground.

"This state of persistent contraction, or rather contracture, of the muscle is not always the same, but varies in intensity, the contraction seeming to be, contrary to what occurs in certain forms of hysterical contracture, in an unstable condition, diminishing and increasing alternately. It sometimes diminishes so much as almost to disappear, and then returns in still greater force. These variations, which often occur spontaneously, may take place under the influence of various causes. The contracture is increased by emotions and by fatigue. It may cease upon the slightest touch, or under the influence of passive movements or massage, but returns again shortly; in spite of the return, however, these manœuvres often give the patient very great and actual relief.

"On examining each muscle closely we see that it is the seat of minute vibrations, the surface being moved by fine undulations which are evidently due to the isolated and successive contractions of the muscular fibrillæ.

"These fibrillary contractions are independent of the tremor with which they are in no way synchronous. But they appear to be the cause of the characteristic rigidity which they maintain by the combined effort of a certain number among themselves; this effort is momentary, but it is incessantly renewed by the contraction of new fibrillæ. The theory of the production of certain contractures, that the muscular fibres contract one after the other, supplementing and following each other incessantly, would be perfectly applicable here."

Finally in the patient who served as the text for Richer's description, the morphological picture was completed by an exaggeration of the subcutaneous venous system, due to the compression of the deeper veins by the muscles. We see the same thing in athletes in whom there is often a dilatation of the superficial veins as a result of the repeated and forcible muscular contractions.

As a rule the muscular rigidity does not become very pronounced until an advanced stage of the disease. The articulations then become fixed in their vicious positions as if soldered there. The rigidity may, in certain instances, precede the tremor or even replace it, this is one of the anomalous forms of the disease.

All writers are agreed that there is a retardation of the voluntary movements in paralysis agitans, but it is not true that this retardation is unaccompanied by any lessening in force of the movements, as Charcot claimed. I have observed in several cases a gradual diminution of motor energy coinciding with a gradual prolongation of the reaction time, and I have found no facts proving the possibility of a retardation independent of a diminished force of the movements.

There are certain peculiarities in the walk of sufferers from paralysis agitans which are interesting to study; there is a tendency to propulsion or to retropulsion. After having slowly and painfully risen from his chair, the patient hesitates to move; then after a few steps his gait becomes more rapid and the length of the steps appears to increase at the same time that they become more rapid; the patient seems to be running after his centre of gravity (Trousseau). Once having started, the patient is unable to stop of his own volition and he would fall unless his progress were arrested by some obstacle. At other times the tendency is to backward motion. If the patient tries to make a step backwards he is forced in spite of himself into a more or less rapid retrograde movement, and he falls unless some one comes to his assistance. This phenomenon may be provoked by slight traction

backwards. Propulsion and retropulsion may exist in the same patient or either one may occur alone. Some patients take an oblique direction in propulsion or retropulsion. Some also have this tendency to lateropulsion in their normal walk. Whatever may be the direction of his impulsive walk the patient always progresses rapidly. In certain cases an analogous phenomenon has been observed of ocular lateropulsion, which interferes considerably with reading (Debove, Neumann).

In a certain number of patients the maintenance of one position is insupportable, and they have an incessant desire to change their attitude or to move to another place. They turn incessantly in bed or if they are sitting they rise every moment to seat themselves again in a different, if not more easy attitude.

The uncomfortable feelings of the patient are still further increased by an habitual sensation of excessive heat; even in midwinter the patients complain of being too warm, and day or night can bear only thin clothing; they are often covered with a profuse perspiration. Sometimes under the influence of a trivial cause they have paroxysms of sweating, or of a generalized burning sensation of the skin. Charcot, who has well described this phenomenon, says that there is no elevation of temperature of the interior of the body. A case has been reported, however, in which there was an elevation of the peripheral temperature of several degrees. This increase of temperature may occur in paroxysms; hot flushes are also frequent in these patients.

Apart from the muscular or rheumatoid pains, which we have seen are occasionally present at the beginning of the trouble, there are hardly any disturbances of general sensibility. There are also no disturbances of the special senses. Galezowski has, however, reported a case of temporary amblyopia; but the ocular troubles which we meet with are specially of a motor order, such as fixity of the regard or falling of the eyelid with paroxysmal tremor.

The exaggeration of the patellar reflexes and the epileptoid trembling which are sometimes seen in aged sufferers from Parkinson's disease may be attributed to spinal lesions associated with arteriosclerosis and do not appear to belong properly to paralysis agitans. The bladder and the rectum are not affected, but the genital functions are, as a rule, early abolished, even in young subjects. There are no trophic disturbances except in the final stages or under the influence of immobility; the muscles then become the seat of atrophy. The general nutrition is affected by the continual loss of saliva, by the abundant sweating, and by the insufficient alimentation.

Bence Jones held that there was an increase in the proportion of

sulphates in the urine in cases of paralysis agitans, but Regnard has obtained the opposite result. Chéron found an increase in the phosphates and regarded phosphaturia as an essential feature of the disease. This phenomenon, which has been found by many observers, has been denied by others of no less authority. As is the case with other neuroses, the chemical formula of paralysis agitans has yet to be discovered.

In the course of paralysis agitans we often observe episodic phenomena which do not seem to belong properly to the disease; such are satyriasis, glycosuria (Topinard), vertiginous, apoplectiform, or epileptiform attacks, which have been attributed to a functional ischæmia caused by temporary spasm of the cerebral vessels.

Ought the psychical troubles which are sometimes manifested in the course of Parkinson's disease to be regarded as troubles belonging to the affection or as accidental phenomena? The polymorphous manifestations, which are chiefly of a depressing nature and sometimes accompanied by an impulsion to suicide, are in no way special to paralysis agitans and indeed are not even common in this disease. Even though they seem occasionally to follow the course of the tremor, we cannot assert that they are associated with it (Ball); it is at least as legitimate to hold that they develop in consequence of the neuropathic predisposition of the patient alongside of the motor troubles, oscillating, like them, with the oscillations in the general condition.

Prognosis.

The course of paralysis agitans is slow and progressive, and it very seldom retrogrades; the most that we can hope for is an arrest in its progress. It is always of long duration, unless the fatal termination comes prematurely through an apoplectiform or epileptiform attack, or, more commonly, a pneumonia to which these patients are peculiarly exposed in consequence of their habit of uncovering themselves at all seasons in order to obtain relief from their sensations of heat. Apart from these accidents, the nutrition is altered under the influence of immobility, diarrhœa appears, bedsores form, and the patient dies in exhaustion.

Diagnosis.

The diagnosis of paralysis agitans is based upon the character of the tremor, on the rigidity, the existence of a constant sensation of excessive heat, the desire to change the position, and the character of the attitude and of the gait.

Multiple sclerosis, which is the disease most liable to be con-

founded with paralysis agitans, is differentiated from it by the character of the tremor, which, instead of being manifested in repose, is produced only during voluntary movements, and is increased in proportion as the hand approaches the object to which it is extended; by the extensive movements of the head, which are exceptional in paralysis agitans, and in any case never very marked; by the nystagmus, diplopia, vertigo, etc.

Senile tremor and essential hereditary tremor, which resemble in many repects that of paralysis agitans, are situated especially in the face, head, and upper extremities; they are remarkable by their isolated character, and also by the fact of their familial character, which is very exceptional in paralysis agitans.

Toxic tremors are differentiated not only by an absence of the characteristics of the Parkinsonian complex but also by the presence of other signs of intoxication, alcoholic, mercurial, etc.

The hemiplegic tremors consecutive to cerebral lesions have a special mode of onset and are accompanied by exaggeration of the tendon reflexes on the affected side (Charcot). Furthermore, the movements in hemichorea, in hemiathetosis, and in cerebral hemitremors differ in their nature from those of hemiparalysis agitans except in rare cases (Grasset).

The tremor of general paralysis of the insane coincides, indeed, with a more or less fixed expression and attitude, a condition of rigidity, at least momentary, and with an habitual sensation of heat, but the disturbances of speech, the psychical troubles, the pupillary phenomena, etc., enable us to differentiate the two affections very clearly.

Hysteria may simulate paralysis agitans, but this simulation is seen chiefly in young women who have, moreover, the stigmata of hysteria.

Treatment.

The disease occurs in conditions of general depression of the organism, and it reposes upon a foundation of depression. The most useful therapeutic measures are precisely those which tend to improve nutrition, namely, hydrotherapy, static electricity, tonics, and massage.

As regards the remedies proposed with a view to calm the most distressing symptoms, their number and variety are a proof of their uselessness. We shall not waste the reader's time in giving the doses of subcarbonate of iron, of hyoscyamine, of iodide of potassium, strychnine, belladonna, ergot, opium, nitrate of silver, arseniate of potassium, bromide of camphor, sulphate of duboisine,[28] solanine,

borax, etc., all of which have been recommended by one or another writer and the value of which is solely as placebos. Francotte, however, has recently stated again that the sulphate of duboisine, in the dose of 2 to 3 mgm. a day, may affect the tremor, at least temporarily.[25]

Charcot noticed that in certain cases an accidental mechanical jolting, such as that sustained in a railway journey, quieted the tremor and caused a certain feeling of comfort. An artificial general trepidation produced by special apparatus, such as the vibratory chair, or the apparatus of Bonnain or of Vigouroux, may give a measure of relief in the same way.

The reduction of the tremor caused by voluntary movements has led to the use of repeated monotonous movements as a palliative measure. Thus Hirt reports a case in which the patient was able to obtain a little rest for his hands and arms by continually turning small objects between the thumb and index finger.

Recently Gauthier,[26] regarding paralysis agitans as a muscular phosphatic dystrophy, has recommended the use of a diet rich in phosphate of potassium and in fats.

THOMSEN'S DISEASE.

The history of this affection dates back scarcely twenty years. Thomsen, who was himself affected with the malady, was the first to describe it, although it had not been entirely unknown before that time. It had been noticed by Ch. Bell, Leyden, and Benedikt. After Thomsen many other writers described it under different names, such as spasmodic spinal paralysis with intermittent tonic contractions of the voluntary muscles (Seeligmüller), muscular rigidity and muscular hypertrophy constituting an autonomous symptom complex (Bernhardt), myotonia congenita (Strümpell), congenital muscular hypertrophy (Jacusiel), muscular spasm at the beginning of voluntary movements (Ballet and Marie). The latter term expresses one of the principal characteristics of the affection. Sometimes the reflex movements of the voluntary muscles are accompanied also by rigidity, which on the other hand never occurs under the influence of passive movements. The smooth muscles are never implicated. When a muscle begins to act we see it stiffen, increase in size, and grow hard. This contraction lasts for from one to three or four seconds and is sometimes accompanied by a special sensation. The rigidity manifests itself only at the beginning of a contraction, and ceases to be noticeable when the muscles have become warmed up to their work. This tendency to rigidity is increased by fatigue, prolonged immobil-

ity, lowering of the external temperature, and by the emotions, especially painful emotions—in some patients it is diminished during the period of digestion. The intensity of the rigidity is variable; sometimes it is extreme. A patient under the care of Benedikt was obliged to get assistance to overcome it and bend the limbs. The lower extremities are often most markedly but not alone affected, and this may give rise to a large number of strange attitudes. The patient under the care of Ballet and Marie, whenever he attempted to mount a horse, felt his left leg stiffen in flexion while the foot was in the stirrup, and then when this had yielded permitting him to raise himself, the right leg became fixed in extension above the horse's crupper. One of Leyden's patients, when he closed his fist, was unable to open it. The tongue may also stiffen and render speech slow and peculiar; the muscles of the eyes may be affected, fixing the gaze spasmodically. The tension of the muscles quite frequently causes a cracking of the joints,[37] not unlike that occurring in hysterical persons.

This tendency to rigidity manifests itself sometimes by a permanent tension and almost constantly by an increase in volume of the muscles. A patient of Seeligmüller had large muscles which, even in a state of repose, were hard as wood and subject to tonic contractions under the influence of mechanical or electrical irritation or of simple blowing on the skin. The strength of the voluntary movements does not correspond to the athletic development of the muscles, but there is, on the contrary, often a notable loss of power. Vigouroux has noted the coincidence of Thomsen's disease and pseudo-hypertrophic paralysis in the same subject. However, not only is the increase of volume and of consistency of the muscles not constant in Thomsen's disease, but in some cases on the contrary disseminated atrophy has been observed. Finally, we sometimes see fibrillary movements, either spontaneous or excited by mechanical stimuli.

Erb has described under the term "myotonic reaction" peculiar modifications of neuromuscular excitability: the response of the nerves to mechanical, faradic, or galvanic excitation is normal or diminished, while, on the contrary, there is hyperexcitability of the muscles to stimuli of all kinds. With the galvanic current we note only closing contractions, as strong at the positive as at the negative pole, torpid, tonic, and rather persistent; these occur except when a current of the very slightest intensity is employed. In many of the muscles strong faradic currents excite undulatory and regular contractions. Stable galvanic currents cause undulations which follow each other in regular rhythm. The myotonic reaction is obtained with special facility in the muscles of the extremities. The effects of local stimulation are often diffused to neighboring muscles.

The condition of the tendon reflexes is variable and in no sense characteristic. There are no constant changes in sensibility. We may observe the coincidence of other nervous troubles of epileptiform or hysteriform character. The mental state varies greatly in different patients. Many are very narrow-minded, and the different forms of morbid emotivity and mental tics are common among them.

Myotonia is, indeed, related hereditarily much more closely to mental affections than to the neuropathic. It is the neuropsychopathic heredity which seems to dominate the etiology of the disease; Thomsen's family was particularly remarkable in this respect. But apart from all neuropathic heredity we often see the disease affect several patients of the same generation; it is particularly a familial disease. Isidore Geoffroy St. Hilaire has remarked that "independently of all hereditary transmission, certain individuals who are perfectly normal (apparently at least) have a sort of predisposition to give birth to abnormal beings, just as others, affected with one or another malady, give birth to well-formed beings, but twins." The teratological malformations are manifested frequently in several children in the same family, although we cannot find any similar defect among the ancestors. There are monstriparæ (Chabry), who produce familial monstrosities, to which familial diseases should be compared." Thomsen's disease falls all the more naturally into the teratological class since, except in rare instances, it is a disease of infancy and often, indeed, appears to be congenital. Besides, although the peripheral nervous system is unaffected (Déjerine and Sottas), there is an alteration in the muscles, first described by Erb, which appears to be constant and which may be compared to arrest of development; it consists in an hypertrophy of the protoplasm and of the nuclei of the non-differentiated substance of the muscle and of an atrophy of the muscular tissue proper. The muscular fibres have an embryonal aspect and functionate like embryonal fibres, the contraction of which is slow and persistent.

The teratological nature of the affection could not be conceded without reserve did not the integrity of the nervous system confirm it. I have shown, indeed, that under the influence of precocious lesions of the nervous system one may see malformations produced which are entirely analogous to the developmental malformations occurring without recognized cause." The insufficiency of post-mortem records explains the diversity of opinion among writers, some of whom regard the affection as one of neuropathic nature, while others consider it of myopathic origin.

Diagnosis.

Thomsen's disease, characterized by a peculiar rigidity appearing at the beginning of voluntary movements, by the myotonic reaction, and by hypertrophy of the muscles coinciding with an actual loss of power, may be differentiated on sight from pseudohypertrophic paralysis, from spasmodic tabes, from intermittent claudication of vascular origin, from localized spasms, or from professional neuroses. It may also be differentiated from the paramyotonia of Eulenburg, characterized by attacks of rigidity followed by more or less complete and more or less lasting paralysis affecting the muscles primarily contractured; by spasm, which is also hereditary and congenital, is symmetrically increased by cold but moderated by heat; and in which the myotonic reaction is absent.

Thomsen's disease, as a rule, progresses by starts, presenting alternations of improvement and aggravation, but always advancing without any prospect of cure so far as has yet been shown. This incurable affection may, however, be compatible with a long life, for it does not affect the general condition.

Treatment.

Among the therapeutic measures which have been recommended are massage and electricity, which can, however scarcely have other than a moral effect. Nevertheless, when muscular atrophy is marked, faradization would appear to be of some service.

PARAMYOCLONUS MULTIPLEX.

Paramyoclonus multiplex, which was first described by Friedreich in 1881, has since been described by various other writers under the names of multiple myoclonus, myoclonia, etc. It is characterized by spasmodic contractions, generally arrhythmical and symmetrical, but not synchronous in the homologous muscles, occurring also at the same time in muscles distant from each other and independent both in nerve supply and in function. These spasms, which are often preceded by sensations of stretching in the muscles, are manifested under the form of clonic, tonic, tetanic, or fibrillary convulsions. The clonic spasms, which are the most common, occur thirty, forty, or fifty times a minute and terminate usually in a painful tetanic contraction; they sometimes occur in series separated by regular intervals; at other times they occur singly. They involve the entire mass of muscle. They occur sometimes in a single muscle, sometimes in several simul-

taneously or alternately, but the muscles affected do not belong exclusively to the same nerve supply; they may even affect all the striated muscles. Most commonly they do not cause any movement of the limb in which they occur, although sometimes there is some displacement correlative to the contraction. The spasms are instantaneous and incoördinate, resembling contractions produced by electricity. All the striated muscles may be affected as well as those of the extremities; the muscles of the face, the tongue, or the abdomen, the diaphragm, and the cremaster may participate in the spasm. Although, as a rule, the contractions are symmetrical they may exceptionally affect only one side of the body; this was the condition in a case described by Chauffard which supervened upon an articular lesion.

The spasms may occur spontaneously during repose, but are more common following effort or fatigue. They cease during sleep and may be moderated for a certain time by an effort of the will. They are made worse by strong emotion, peripheral irritation, percussion of the muscles, or irritation of the skin. The influence of position is very remarkable. Lying in bed in the horizontal position increases the frequency and intensity of the spasms or provokes paroxysms. In the sitting position the spasms extend to the members and to the head, but the trunk preserves a relative immobility. When the patient stands the lower extremities enjoy a relative calm; during walking the spasms are suspended or at least so attenuated as not to interfere with progression. Speaking generally, voluntary movements have a restraining effect upon the spasms, but voluntary movements executed during one of the attacks are usually painful.

The tendon reflexes are often exaggerated. The electrical reactions are normal. The sphincters are not affected. In exceptional cases vasomotor troubles are observed, such as cyanosis of the extremities and profuse sweats (Seeligmüller), but as a rule there are no trophic disturbances. Sensation in all forms remains intact. Besides the muscular pains there are sometimes pains in the back and wandering pain in the limbs. The intellect is usually unaffected.

The progress of the disease is insidious. The spasms are ordinarily preceded by dull pains in the limbs and a sensation of general weakness. In consequence of some emotion, of a traumatism, or of fatigue the patient is seized with muscular contractions, which affect exclusively the voluntary muscles. These begin in the muscles of the lower extremities, the function of which they do not usually affect; little by little they extend, at the same time their intensity increases, and then interference with function is manifested. There are often periods of recrudescence under the form of crises during which the convulsions have sometimes the energy of choreic spasms. Some-

times the sensation of general weakness occupies for a long time a predominant place in the symptomatology.

The duration of the affection may be very long. It may remain stationary during many years. It is susceptible of amelioration, even of cure, but relapses are common when the patient exposes himself to the original causes.

The etiology of paramyoclonus multiplex is still obscure. In rare cases an hereditary influence has been noted (Gucci). It occurs ordinarily in conditions of general debility, following a mental or physical shock, overwork, or an infection like that of diphtheria. Lemoine and Lemaire regard it as allied to neurasthenia.

In certain cases a cure has been attributed to the application of the galvanic current along the spinal column, but we must not forget that there are cases of spontaneous recovery. We ought to attend with great care to the general condition.

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NEURASTHENIA.

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NEURASTHENIA.

History.

THERE is no doubt that some of the symptoms which we at the present time recognize as "neurasthenic" have existed since civilization began, and in particular since the time when people lived in cities where the stress of competition and the opportunities for luxurious living are great. There is, however, very little positive evidence of anything like neurasthenia in ancient or classical history. It has been suggested that the Greeks suffered from neurasthenia because Alcibiades had palpitation of the heart in the presence of Socrates, and that there was neurasthenia in Rome because Augustus had a complaint which was cured by hydrotherapy. But this is hardly evidence. It is a fair inference, however, that the Romans, who showed in their imperial families such marked signs of mental degeneration, must at the same time have suffered from those milder conditions which are represented by neurasthenia. They did not, however, have to contend against the sinister effects of syphilis, tobacco, tea, coffee, railroads, electricity, and daily newspapers.

In 1540 the celebrated clinician Fernel wrote a work, "De Abditis Rerum Causis," in which he ascribed various hysterical and nervous symptoms to "vapors." These were produced, as he thought, by a decomposition of the sperma or menstrual blood, which then passed up through the system, disordering the nerves. In this way arose the clinical term "vapors" as expressive of hysterical and hypochondriacal states.

In the seventeenth century medical writers first began to describe at length symptoms that were then called "the vapors," "hypochondriasis," "the spleen," and the "nerve sickness" (*maux des nerfs*). The credit of first separating out the class of nervous disorders from hysteria and hypochondriasis is given by some to Dr. Robert Whytt (spelled also Whyte), who published a book on "Nervous Disorders" in Edinburgh in 1765. Dr. Whytt said that the persons suffering from nervous disorders could be divided into three classes, the nervous, the hysterical, and the hypochondriacal. It is very evident, however, from his description of the "nervous" class that he did not

come in contact with anything like modern neurasthenia. Nervous people, according to his idea, were those who were affected with "tremors, palpitations, faintings, and convulsive fits," and this is the limit of his symptomatology.

Dr. George Cheyne wrote a book entitled "The English Disease," published in 1785, in which he gives a very good description of what at that time was called "hypochondriasis," but which corresponds fairly well to what we would now call "lithæmic neurasthenia." His patients were evidently, however, all sufferers from idleness and luxurious living. In the early part of the nineteenth century there were many further contributions to this same subject. The one which perhaps made the most mark in clinical medicine was that upon "Spinal Irritation," by the brothers W. Griffin and D. Griffin, published in 1834. Under this name these authors described a very large number of symptoms which are now classed under the head of neurasthenia. They, however, made the serious mistake of trying to fit all the symptoms to a theory which placed the morbid condition in relation with the sensory nerves of the spine.

The first writer to bring forward in a particularly distinct way the neurasthenic symptoms, and to separate them from hypochondriasis and hysteria, was Dr. E. Bouchut, whose work upon the "Nervous State in Its Chronic and Acute Form" was published in 1857 and 1858. A revised edition was again published in 1879, under the title of "Acute and Chronic Nervousness and Nervous Diseases" ("Du nervosisme aigu et chronique et des maladies nerveuses"). Bouchut included under the designation nervousness or *nervosisme* a series of morbid phenomena which had heretofore not been regarded as representing a special disease, but rather only as morbid conditions, and these morbid conditions had been previously considered, either as part of hysteria or of hypochondriasis, or of some of the psychoses. Bouchut urged that this *nervosisme* was a disease *sui generis*, like hysteria, epilepsy, or melancholia. He considered it a functional trouble, and divided it into acute and chronic types.

The word neurasthenia is to be found in Dunglison's "Medical Dictionary," published in 1833. But in 1867 Dr. E. H. Van Deusen, of Kalamazoo, Mich., first used the term for purposes of clinical description (Supplement to the biennial report to the Michigan Asylum for the Insane). The title of his article was "Observations on a Form of Nervous Exhaustion Culminating in Insanity."

Next in the chronological succession, and undoubtedly first in importance in connection with the history of neurasthenia, stands the work of Dr. George M. Beard. Dr. Beard was born in Montville, Conn., in 1839, and died in New York City in 1882. His earliest

contribution to the subject was published in 1869 in the Boston *Medical and Surgical Journal*, ten years before Bouchut's work appeared under its later title. Dr. Beard's most important works were his book entitled "Neurasthenia: Its Symptoms, Nature, Consequences, and Treatment," published in 1880, and another work in which his views were still more amplified, entitled "American Nervousness" (1881). He published in all three books and twelve or more short articles upon this subject. Dr. Beard deserves the credit of calling the attention of the profession to the separateness and importance of the neurasthenic state. Through his acuteness of observation and originality of mind many symptoms were put on record which had been previously unrecognized or ignored. Despite an enormous amount of prejudice and opposition, he forced the medical profession to see that there was truth in his views, and though he died before he accomplished all that he hoped, he left behind work which made for him an enduring monument. At the time of Dr. Beard's death there was no other treatise in the English language which deigned even to mention in any systematic way the subject of neurasthenia; although the Germans had translated his book, giving him credit for originality; and German as well as French clinicians of the highest rank, including Erb and Charcot, had adopted the term "neurasthenia," and acknowledged the essential soundness of his views.

Dr. Beard showed that the large class of symptoms which had been previously referred to hypochondriacal fancy, to disease of the stomach, disease of the uterus, perverted conditions of the liver, and urinary excretions, were really and fundamentally dependent upon a morbid weakness of the nerve centres, and the result of his writings and the propaganda which he started is that now practically all medical men of experience agree that there is a morbid condition of which the underlying cause is a nervous irritation or defect.

Definition.

Neurasthenia may be defined as a chronic functional nervous disorder, which is characterized by an excessive nervous weakness and nervous irritability, so that the patient is exhausted by slight causes, and reacts morbidly to slight irritations.

Etiology.

Some doubt has recently been thrown over the question of the excessive nervousness of the civilized nations of the present time. It is not a matter which can be fairly settled by statistics or the perusal of historical documents, but, on the whole, the evidence is, to my mind, conclusive that the human race does now suffer relatively

more from nervous irritability and exhaustion, in its various types, than it did in the past. This I infer partly from the fact that the predisposing and exciting causes of neurasthenia are more largely present now than they used to be. The tendency of people to city rather than rural life is perhaps one of the strongest points in favor of this view, since we know it is in our urban population that neurasthenia breeds best. A larger proportion of persons now also use their brains in the struggle for existence and live upon a higher mental plane, with all the danger which that implies. The eighteenth century writers attributed all the functional disorders then known, under the terms "vapors," "spleen," "hypochondria," "hysteria," to three things—luxurious living, sedentary life, and the unsanitary conditions of great and populous cities (*vide* Cheyne, *op. cit.*). They said nothing about the effects of overwork, continual anxiety, and mental strain, and one certainly does not gain from reading the English medical literature of this period that there was any such excess of work and worry among the people.

Without going into any further argument upon this point, I shall admit, as an offset to this, the more widespread knowledge of how to live and how to ward off disease, so that it is at least probable that even if neurasthenia is more prevalent now, and has been steadily increasing, this will not necessarily always continue to be the case.

At the present time we know that neurasthenia is found more frequently among the highly cultivated races. I have seen it in negroes, but it is extremely rare, while hysteria and insanity are fairly common. I believe that Americans deserve to an extent the reputation which they have of suffering greatly from neurasthenia. This is particularly the case in the Northern and Central States, on the Colorado plateau, in parts of California, and in the great cities of the East. Neurasthenia is said to be quite prevalent in Russia, and it is generally observed that it affects particularly often the Hebrew race. In this country we see it quite often in the Irish, but almost as often in the English, and rather less frequently in my experience in the Germans.

Neurasthenia prevails rather more in dry temperate climates, but it is by no means infrequent in the tropical regions, and is to be found in the West Indies and in the republics of South and Central America in its classical forms.

Neurasthenia is found rather more often in men than in women, but the difference is not great. Among 828 neurasthenics whose histories were analyzed by Hösslin, there were 604 men and 224 women, but this does not give the proper ratios if we include all grades of society.

The neurasthenic age ranges from eighteen to fifty-five, but the larger proportion of cases is met with between the years of twenty and fifty. Occasionally symptoms resembling neurasthenia may be seen in children of the age of twelve or thirteen, and occasionally also there develops a kind of senile neurasthenia, which is, however, often associated with hypochondriasis, and some definite degenerative changes in the nervous or vascular system.

In men neurasthenia occurs more often in the single; in women the relation is somewhat reversed, so that, taking both classes, the married and the unmarried are about equal.

Neurasthenia does not much affect the people of the country and small towns, though it does exist there. In great cities the number of neurasthenic women, among the wives of laborers and artisans, is rather large, and this is the natural result of the strain of living with husbands who are dissipated, and of rearing large families of children in the close quarters of a tenement house. The disease is relatively more frequent in the educated classes.

Hereditary influence plays a considerable part in the development of neurasthenia. We can usually find that there is a history of migraine or some nervous irritability upon one side or the other. Quite often there is also a history of rheumatism or gout or of tuberculosis. A distinct history of the major neuroses or of severe mental diseases is rare, but still it is my belief that a very large proportion of neurasthenics come into the world with an over-sensitive and weakened nervous system. They may be strong enough to undergo the ordinary strain of life, but break down under some specially exciting causes. Hösslin found among his eight hundred and twenty-eight neurasthenics positive evidence of a bad nervous heredity in thirty-five per cent.

The exciting causes of neurasthenia are very various, but they can most of them be classed under the head of excessive mental strain or shock, sexual abuse, and the influences of exhausting fevers, of chronic infections like syphilis, and of poisoning with alcohol and tobacco or tea and coffee. In the larger proportion of cases of men, the trouble if it develops during adolescence is brought on by overwork at school and in college, combined with neglect of sleep and carelessness in diet. Frequently the abuses of the sexual function, of tobacco, or of athletics are the exciting causes.

The practice of masturbation is one of the things for which neurasthenics very often keenly reproach themselves and over which much hypochondriacal brooding develops. Excesses of this kind, however, are usually a sign of a degenerate or unbalanced nervous system rather than a cause. The actual harm done is greatly exag-

gerated, however strongly this practice is to be reprobated. Excessive and unnatural indulgences, such as sodomy, etc., tend to weaken the nervous system and are causal factors of neurasthenia. Bad methods of education and in particular excessive study are thought to predispose to nervous exhaustion. This is usually seen in ambitious college students, or in young men who are forcing their way under great disadvantages through professional schools and into professional practice. Young women, who are excessively devoted to study and yet cannot refrain from social indulgences, sometimes break down with nervous exhaustion. The studies and training of the primary and secondary schools may prepare the way for these catastrophes, but they rarely come before the eighteenth year.

Typical attacks of neurasthenia are undoubtedly brought on by the fright and shock incident to severe injuries or exposure to great danger, as in railroad collisions and other frightful forms of accident. A large proportion of the so-called "traumatic neuroses" are simply forms of neurasthenia.

Neurasthenia can be brought on also by excessive child-bearing, the drain of lactation and domestic trouble, great excesses in eating and drinking, and the strain of hard domestic life and of sickness and nursing.

Neurasthenia sometimes follows an acute infection like that of typhoid fever or the grippe. It also may be induced by the infection of syphilis. It then comes on in the secondary or less often in the tertiary stage (Fournier). It is probable that in many of these cases the trouble is due to the excessive use of mercury and saline purges. At any rate antisyphilitic treatment can certainly bring on or bring out a neurasthenia. A combination of secondary syphilis with the excessive use of alcohol leads to a very obstinate type of neurasthenia. Malarial poisoning seems also to have some influence as the exciting cause. Much weight has been laid upon the importance of eye strain in producing neurasthenia, and, given a neuropathic constitution, there is no doubt that the defects in the refraction of the eye or in muscular equilibrium may cause, or at least keep up, a neurasthenic state. The same is probably true of severe forms of gastric disturbance, and of disease of the pelvic organs, such as subinvolution, decided displacements, and chronic ovaritis or salpingitis. In men the existence of prostatic irritation, of irritable strictures, and hemorrhoids and fissures, may start up neurasthenic symptoms. Chronic middle-ear disease and nasal stenosis are also put down as occasional exciting causes.

The existence in neurasthenics of a tendency to constipation, or what is popularly known as "biliousness," accompanied with a gouty

or lithæmic diathesis, has been much dwelt upon, and at one time neurasthenia was thought to be largely the expression of a disturbed state of the metabolism—a phase only of gout or lithæmia. I am convinced, however, that this tendency is rather the result of the weak nerve centres than the cause, though the two often act in a vicious circle. Prolonged and severe dyspeptic disturbances, especially when associated with atony of the stomach and bowels and the condition known as enteroptosis, are exciting or maintaining causes.

I would sum up the leading causes of neurasthenia thus:

1. Hereditary nerve sensitiveness.
2. Overwork and worry.
3. Severe shocks, with or without injury.
4. Infections.
5. Abuse of stimulants and narcotics.
6. Abuse of sexual functions.
7. Abuse of digestive functions.

This means that the causes are most often a bad heredity and foolish living.

A great deal of stress has been laid upon autotoxæmia as a cause of neurasthenia. There is no doubt that a great many symptoms and crises are brought about through this agency, but the attempts to prevent autotoxæmia by perpetually stimulating the liver and giving intestinal antiseptics, of using a large amount of water and the simplest kind of diet, do not of themselves cure the disease, unless measures are taken to strengthen the impaired tone of the general system.

Symptoms.

The symptoms of neurasthenia, while manifold, have yet a pretty distinct general resemblance to each other, and the clinical picture of typical forms of neurasthenia is quite as pronounced as that of other nervous maladies. The patient's symptoms are to be sure nearly always of the subjective character, but they are reiterated with so much force and feeling, and the independent descriptions tally so closely, that one can hardly fail to be convinced by the story itself that they are expressions of the same morbid condition.

The patient complains of a general feeling of mental depression—life is not the interesting spectacle to him that it formerly was. The man who once delighted in work can hardly force himself now to go to it. He tires very quickly over tasks which were formerly easily performed. He loses his power of originating plans and of mapping out work. He absolutely cannot pursue a train of thought or a single line of work for a long time, but sits idly at his desk or

goes back to his home in depression and despair. He is very easily irritated at things which before caused him no annoyance, and becomes a source of domestic unrest and unhappiness. He is oppressed with the fear that he will never get well, or is going to become insane or paralyzed, or that some dreadful termination of his present malady is bound to occur. He sleeps badly, waking up perhaps after a short rest in the early part of the night, or, if he sleeps until morning, he has disquieting dreams, and wakes up unrefreshed. He suffers from a number of peculiar sensations which are called "cephalic paræsthesiæ." These are sensations of pressure on the top of the head, or a feeling of constriction around the temples, or a burning spot on the vertex, or tenderness of the scalp. Sometimes he has a sense of weakness or even pain in the back of the neck. He has also peculiar paræsthesiæ of the hands and limbs; they feel numb or asleep at times. Peculiar chilly sensations creep up the back or legs. He less often has attacks of dizziness; spots come before the eyes and buzzing sounds are heard in the ears and head. Headache occurs in perhaps one-half of the cases, the headache being usually either frontal or occipital. It is often very persistent and in fact a chronic headache, not due to tumor or meningitis or syphilis, is almost invariably of neurasthenic origin. This neurasthenic headache is usually diurnal only, coming on in the morning when the patient wakes up, and lasting a good part of the day. It does not often keep him awake at night. In this point it is distinguished from the headaches of syphilis and of meningitis or of tumors. Women suffer from these headaches, and from pains in general, more often than men. They in particular have much pain in the back of the neck and along the spine. This keeps them from walking or being upon their feet, and it may develop into a form of neurasthenia known as "spinal irritation."

The special senses are not very seriously affected. The patients can often see quite well, but their eyes soon tire; the effort of watching a play fatigues them. They cannot read a book long because it makes the eyes smart or produces some headache. Examination of the neurasthenic's eye frequently shows the existence of some refractive error, most frequently astigmatism and hypermetropia; defects in the ocular muscles, and especially weakness of the internal recti, often occur. Patients have frequently complained to me of a defect in visual memory. They see a thing or face but do not remember it again as readily as they used to. There is no limitation of the visual field in true neurasthenia uncomplicated by organic disease, but there is a morbid susceptibility to fatigue, particularly of the periphery of the vision, so that, after long testing, objects in the periphery

become less distinct, and a sort of artificial limitation of the field may be produced. In some cases an object which is brought from without into and across the visual field is seen in wider range than an object which is placed in the centre of vision and carried gradually out towards the periphery. This is the reverse of the normal condition, and is known as "Foerster's shifting type." Peculiarities of accommodation, a slight drooping of the lids, inequality of the pupils, and excessive mobility of the iris have been noted in neurasthenia.

As I have already stated, neurasthenics sometimes suffer from tinnitus, which is very distressing and aggravates every other nervous symptom, but this usually occurs only in connection with actual disease of the middle ear, or in old people with degenerative changes in the cerebral blood-vessels. An excessive sensibility to noises, and even to pleasant sounds, like those of music, may be present. Neurasthenics sometimes cannot bear even the most enchanting melodies. A similar morbid sensibility to taste and smell may be present. But these are matters of minor moment, and are much more often seen in hysteria or in a hystero-neurasthenia.

There is no doubt in my mind that in neurasthenics the general muscular and nervous strength is lessened, and although the patient may not have lost flesh, and may not appear particularly weak, he tires quickly on ordinary exertion, and the tests of the dynamometer show a lessened response. A fine tremor of the hands is often present, and when the eyes are tightly closed there will be a quivering of the lids, and in very acute and exaggerated cases, twitchings of the muscles of the face and tongue, almost like those in general paresis. This rarely occurs, however, unless the patient has, in addition to the neurasthenia, a considerable amount of toxæmia from alcohol, tobacco, or tea.

The reflexes are exaggerated very greatly. In many cases a blow upon the leg, anywhere from the patella to the middle of the shinbone, will bring out a prompt reaction, and similarly a blow struck on the thigh anywhere from the patella up half-way to along the thigh, will produce a knee jerk. And blows upon the motor points promptly bring out responsive contractions. The cutaneous reflexes are also exaggerated. These things vary considerably, however, in different cases, and are more marked in the younger patients and those of a neuropathic constitution.

The sexual function is irritable and weak.

Lehr has observed in one hundred and sixty-five neurotics a functional disturbance of the heart's activity one hundred and three times. This corresponds with the general experience that there is a considerable disturbance of the heart function in neurasthenia. The most

frequent condition that I have seen is an acceleration of the pulse beat from very slight cause, due to a weakening of the inhibition of the heart. A pressure over some painful point in the body will sometimes bring up the pulse from 80 or 90 to over 100, and it will remain there for one or two minutes. This is called "Rumpf's symptom." Arrhythmia and palpitation of the heart are less frequently observed. It is my belief that cardiac weakness is an important condition in many forms of neurasthenia, and underlies sometimes a good many of the other symptoms. This is particularly true of the neurasthenias of more advanced life. The cardiac disturbances are more frequent in women, in young people, and in neurasthenia associated with the use of tobacco and tea.

A great deal of emphasis has been laid upon the vasomotor disturbances of neurasthenia, and a large number of neurasthenic symptoms have been ascribed to a weakening of the vasomotor centre. As a result of this the patient suffers from cold hands and feet, from flushing of the face alternating with pallor, from dermographic skin, and from those symptoms which we usually attribute to cerebral congestion, such as a sense of fulness in the head, headache, spots before the eyes, dizziness, and noises in the head. Sphygmograms of the pulse show a lowering of arterial tension, and perhaps still more a great variability in the tension of arteries.

The condition of the urine has been studied very closely in connection with this subject. In fact many of the symptoms which we now call "neurasthenic" were described by Dr. Prout and Dr. Golding Bird early in the century and were held by these gentlemen to be due to oxaluria. This was a condition characterized by flatulent dyspepsia, melancholia, and nervous irritability, and was thought to be due to defective metabolism, resulting in the production of an excess of oxalic acid. More recent studies have shown that oxaluria is only one of the manifestations of lithæmia, and that while it is significant, as was then supposed, of defective nutritive changes, these are more dependent on a neurasthenic state than primarily upon dyspepsia and metabolic disorder. There are, according to Herter, few cases of neurasthenia which do not show in the urine or fæces some indication of defective metabolism. "The fæces often contain excessive amounts of urobilin or some related substance. The urine is usually concentrated and of small volume (600–1,000 c.c. in twenty-four hours). Frequently there is an excessive excretion of phosphoric acid (P_2O_5) and an alteration in the quantitative relation of urea and uric acid. In health the relation of the uric acid to the urea excreted varies between 1 to 45 and 1 to 60 in adults. In neurasthenia (as well as some other conditions) the relation is often 1 to

40, 1 to 35, or 1 to 30. Indican is often present in pathological quantities, especially in cases of sexual neurasthenia. Oxalate of lime is often present in excess in the urine." ("Diagnosis of Nervous Diseases," p. 547.)

In rare cases one finds in neurasthenics a temporary albuminuria; I have observed it only once in 100 cases. Hösslin, in 822 neurasthenics, found albuminuria three times. This albuminuria is not associated with the presence of casts or other evidence of kidney disease, and it is apparently due to a paresis of the vasomotor nerves of the kidneys. Transitory glycosuria is more often found. This glycosuria is usually associated with a heavy urine and evidences of lithæmia. Some authors (Hösslin, Dercum) state that there is an excess of uric acid very uniformly in neurasthenia, and that this uric acid results from a breaking-up of the nuclein of the cells. It has been ingeniously suggested, therefore, that since the nuclei of nerve cells become smaller and irregular in shape when the cell is exhausted, it is from this source that the uric-acid excess comes. It is to be remembered, however, that only a small percentage of the total nuclein of the body is in the nerve cells, and it is not easy to understand how any excessive fatigue of these cells could materially increase the amount of uric acid, if the other tissues of the body were in normal condition. As a matter of fact, all observation shows that excessive use of nervous tissue leads to an excessive excretion of phosphoric acid rather than of the urates.

My experience in studying the urine of neurasthenia is that in the younger cases, with a strong neuropathic taint, it is variable in specific gravity, but, on the whole, rather low; and that the daily amount, as Dr. Herter states, is below the average. It is of a low specific gravity also in neurasthenia, occurring after middle life, when the arterial changes of that period begin to set in. In early adult life the urine is more often found to be condensed, as others have observed, and to contain the products of defective metabolism. The urine thus shows either a weakened and slowed-up nitrogenous metabolism or a perverted metabolism. The important things to determine, then, in examining the urine, after excluding such evidences of serious change as albumin and sugar, are: the specific gravity and the daily amount, the amount of phosphates, the amount of urates and uric acid and their relation to each other, and finally, the presence of indican or other products of perverted nutrition and digestion. I do not find indican very often, and practically never in the light urines.

The digestion of neurasthenics is often more or less affected, and a large proportion of them are probably treated mainly for their

stomach conditions. I do not, however, usually find cases of s
and genuine gastric disturbance. In the majority, under
treatment and proper diet, the tongue soon cleans up, and the
complains relatively little of the stomach, though his nervous
toms continue. The neurasthenic, it is true, has always a
digestion, and has to take great care of what he eats and drin
when put upon the kind of diet that he should take, the stomac
relatively little trouble. The common form of dyspepsia
which is associated with acidity, flatulence, some epigastric
ness, and constipation. The tongue is often furred, there is
agreeable taste in the mouth, and frequently anorexia. It is
patients who have abused themselves with alcohol or tobacco
cessive indulgence in sweets, or with ravenous feeding, that
conditions are found. In people of more advanced age, ho
feebleness of digestion is often associated with a relaxation
stomach and intestinal walls, and a great deal of atony of the
intestinal tract. In these cases, which we find particularly o
women, there may be a weakness of the abdominal walls, an
it a certain amount of prolapse of the large bowel and stomac
a great many distressing symptoms resulting therefrom. Thi
dition has been described by Glénard under the name of "e
tosis," and it undoubtedly is an important factor in keeping
neurasthenia of some women in adult and middle life.

Among the most serious, though fortunately rare, sympt
neurasthenia, involving the digestive tract, is the condition
as "mucous enteritis." This trouble generally attacks women
than men, and usually women between the ages of twenty-five and
It comes on after the patient has become exhausted by prolong
mestic cares and fashionable dissipation, or some shock. It
of the earlier symptoms of the nervous weakness, and begin
abdominal pain, followed by attacks of diarrhœa, in which t
casts are passed, or portions of such. This diarrhœa is p
colicky, and alternates with periods of constipation. There is,
experience and in that of others, a somewhat spastic condition
bowel, as though it were irritated and closed down upon the co
of the intestine. The term *mucous enteritis* is not strictly a
one, since microscopical examinations and autopsical reports
that the substances thrown off are not mucous mainly, and tha
is no actual inflammation. The casts that are found in the sto
composed principally of albuminous substance, the product
ently of the decomposition or disintegration of the epithelia
of the intestinal wall. While mucous enteritis sometimes occ
persons who are profoundly asthenic without any decided n

thenia, yet, in the great majority of cases, it is a symptom of neurasthenia, and can only be successfully treated on such a basis.

I do not attempt here to go into a description of the very large number of other symptoms which the industry of specialists has associated with gastric neurasthenia. They include the whole range of anæsthesias, hyperæsthesias, and paræsthesias, as well as motor and secretory disturbances of the alimentary tract. As already stated, I do not in my own experience find that the condition of the digestion is a serious obstacle to securing relief and cure in neurasthenics. There is no doubt that the liver, like the other organs of the body in neurasthenics, acts feebly, and that it is from this cause that many of the products of defective metabolism get into the system. The blood absorbs the imperfectly digested and metabolized products, and thereby causes many of the peculiar neurasthenic symptoms.

The respirations in neurasthenia are generally normal, but shallow and deficient respiratory expansion sometimes exists. In women particularly I have often found that there was an actual inability to properly expand the chest and inflate the lungs.

The temperature is normal, and a very variable temperature of the skin is simply dependent upon vasomotor instability.

The composition of the blood is often quite normal. Hösslin finds that even in those patients who appear to be anæmic there is a normal amount of hæmoglobin; however, anæmia certainly exists in many cases, and there is no question that the use of iron is often of great benefit.

Variations in the weight of the neurotic often occur. Neurotic patients may gain or lose ten or twenty pounds within a year or two. The secretions of the skin are usually increased, and the patient sweats easily and profusely. In other cases of a less irritative type the skin is inclined to be dry. Its nutritional condition is poor, the hair falls; and according to Beard there is a tendency to early decay of the teeth.

The foregoing description of the general symptomatology of neurasthenia is likely to confuse the reader on account of the multiplicity and wide extent of the symptoms. It is quite true that few neurasthenics have all of the symptoms just described, and it is still more true that in most of them the patients have certain leading and dominant symptoms which annoy and depress them, and that the larger proportion of the manifestations of the disorder are trivial to them, as they are to the physician.

The Different Forms of Neurasthenia.

The peculiar type of neurasthenia depends mainly upon the age, the sex, and the hereditary endowments of the individual.

Neurasthenia appearing at the time of adolescence is much more apt to be associated with a primarily weak nervous constitution. The mental symptoms are mainly dominant, and the malady takes more often the character of a hypochondriasis, with some fixed ideas, or morbid fears, such as suggest an incompletely developed paranoia. Naturally, also, at this time, sexual ideas and sexual symptoms very largely predominate. In women neurasthenia at this period is often associated with hysteria, and the French term *hystero-neurasthenia* is frequently a very apt one for the condition. In other cases, women suffer from a great deal of pain along the back, and that particular condition, known as "spinal irritation," complicates the neurasthenic state.

It is during the mature, active life of men and women that the more typical forms of neurasthenia occur. In these patients the element of heredity is less marked, while the extrinsic causes of neurasthenia, such as excesses in eating and drinking, shocks, injuries, poisons, syphilis, and gouty tendencies, all come much more into play.

In neurasthenia developing in middle life and at the period of the climacteric, the disease is associated with the natural symptoms that come from beginning degeneration of the arteries and a diminished resistance of the body generally. There is a greater physical weakness, and we often see at this time, also, neurasthenia associated with much vasomotor disturbance, or with the psychoses, such in particular as melancholia.

Special Types of Neurasthenia.

It is useful to separate certain of the symptoms of neurasthenia into sub-groups. I shall therefore describe briefly some of the specially well-marked symptom groups which the clinician observes.

Traumatic Neurasthenia.

After receiving an injury which is often but slight, but which is usually accompanied by a great deal of fright and emotional disturbance, the patient goes to his home feeling perhaps a little nervous and shaken, but not suffering to any great extent. He goes to bed and sleeps; he wakes up the next morning feeling not quite so well as usual, but congratulating himself, perhaps, on having gotten off

so easily. He resumes his work and finds that he can do it, though with not quite so much ease as usual, and he very likely suffers from some pain due to a strain or bruise that he has received. In a few days—almost always within a week—he begins to notice that he is more nervous than usual, that little things irritate him which did not do so before, that his head seems somewhat confused, and that the effort to work is wearying. His sleep is disturbed, and he wakes up in the morning unrefreshed by his night's repose. He becomes somewhat despondent over his condition, and thoughts of paralysis or some other serious ailment annoy him. His head aches, the pain being more or less constant and diffused, and located usually over the forehead or at the back of the neck. He has unpleasant sensations in the head, such as that of constriction or pressure or scalding feelings. His back also is continually painful, and walking increases it. His nervousness becomes more marked, and close examination shows a little, fine tremor in the hands. He has also sometimes creeping sensations over the body or numb feelings in the extremities. He tires very easily. He is emotional, and becomes more despondent as the days go on. Sometimes he has spots before his eyes, noises in his head, or ringing in the ears. Reading is laborious and increases his headache; so also does attention to work. His appetite becomes capricious and his bowels are constipated. He suffers somewhat from flatulency and dyspepsia. His heart palpitates easily, and the pulse is a little accelerated. Sometimes for a few days there is a little weakness about the bladder or irritability of that viscus. His sexual power is diminished; his circulation seems rather poorer than usual. Very slight excitement produces sweating of the hands or coldness of the extremities. He loses a little flesh.

These symptoms may be several weeks in developing, and during this time he may perhaps consult a lawyer about his case. If so, the anxieties of litigation begin to add to and intensify his troubles. He consults a physician, and the physician finds the subjective symptoms that I have mentioned. Objectively, when examined, the physician will discover that the muscular power is somewhat weakened, that there is a certain amount of fine tremor perhaps in his hands. The knee jerks and elbow jerks are exaggerated; there are tender points along the spine and upon the head. On making him stand with his eyes closed there is a certain amount of static ataxia discovered. The pupils are often dilated and mobile, and examination of the visual field shows sometimes a slight contraction, at other times the "shifting type" already described. In many cases a degree of peripheral retinal anæsthesia will be discovered. The pulse will be found accelerated, and pressure on a tender point may send it up very rapidly;

a slight exertion will also accelerate it. There will be something apparent in the physiognomy of the case which shows the man to be in a nervous and asthenic condition. Sometimes the pains from which the patient suffers in the back and the weariness in the limbs are so great that he remains a good deal of the time in bed. In all cases he will assert most positively that he is unable to work or to take that interest in his affairs that he has previously done. In a good many cases there will be added to the foregoing picture a number of symptoms due to some local injury; for example, the arm may have been wrenched or bruised, and the result may be a certain amount of neuritis and weakness or pain in that member; in other cases the back may have been so severely sprained that the typical symptoms of spinal irritation ensue, and this is particularly apt to be the case when women are injured. In other cases, again, the legs may have been hurt to such an extent that a sciatica or some other form of neuralgia develops.

The foregoing symptoms, varying in amount and degree, will last, with little change, for a very long period of time. If the case goes into litigation, there is added the worriment occasioned by having to go through the disturbing experiences of trial by jury. In many cases, after the trial has been settled and damages awarded or otherwise, the patient begins to mend, and in a certain proportion of cases he gets completely well. This is not invariably the rule.

Spinal Irritation.

Spinal irritation is a form of neurasthenia in which, associated with the general neurasthenic symptoms, are certain special, painful symptoms, related chiefly to the sensory nerves of the spine. These cases have in the past been described under the head of "spinal anæmia" and "hyperæmia." They may develop in traumatic neurasthenia. The patients are usually young women, between the ages of sixteen and twenty-five. The trouble is sometimes brought on by injuries or by a physical overstrain. Sometimes it seems to be associated with a natural weakness of the spinal muscles and a consequent curvature. Sometimes it follows acute infectious diseases. The patient begins by complaining of pain in the back—usually in the lower part—and also in the back of the neck. These pains occur on standing or walking, or any exertion, and are so severe that the patient in the course of a few weeks or months gives up attempting to walk about. They get relief and comfort in bed, and so they go there and remain. The pains are of a heavy, aching character, increased until they become very sharp when attempts at movement of the trunk are made. There is a great deal of tenderness to pressure along the spinal proc-

esses, some of these processes being much more sensitive than others. The most sensitive points are usually in the back of the neck and the upper dorsal vertebræ, and down in the lumbar region. There is some pain also upon pressure alongside of the spinal processes. Painful points often vary, and even in a single examination the patient may complain, and complain honestly, of different sensitive vertebræ. Pressure on these points does not often bring out visceral symptoms, as the brothers Griffin taught. The patients suffer much from headaches. The arms are often weak, so that attempting to sew or write or hold a book causes pain in the neck and shoulders. The legs are also weak and the circulation is poor. There is sometimes palpitation of the heart and precordial distress. A certain amount of dyspepsia is always present, and constipation is the rule. The patients often have attacks of vomiting, and attempts to feed them require much care. The menstrual functions become irregular. The patient grows weaker and often becomes bed-ridden, especially if little attempt is made to overcome the symptoms by voluntary effort and attention to nutrition. These patients generally get well in one to three years, but occasionally they sink into permanent invalidism.

The Anxiety Neurosis, or Neurasthenia with Fixed Idea.

Sometimes neurasthenia is associated with some single idea that becomes fixed in the mind, and worries and harasses the patient through every moment of the waking hours. This idea is always one of a depressing character, and usually one associated with either remorse or fright. For example, one patient of mine had for one or two years an ordinary type of neurasthenia, with a simple nervousness, depression, insomnia, and cerebral paræsthesia. After being well for a few years the trouble returned, this time with a fixed idea that during her early life she had committed a very wrong act. The act itself was a trivial one, connected with the taking of a dose of medicine to bring on her courses. But no amount of assurance could entirely relieve her from the distress caused by the continual presence of this remorseful idea.

In other instances a patient will have a neurasthenia following some severe domestic calamity or some shock or injury. Associated with the general neurasthenic symptoms, may be an intense fear that she is going to die from some heart trouble, and the patient is continually running to her physician, and feeling of her pulse, under the apprehension that she may drop dead. This condition is not one, strictly speaking, of hyponchondriasis, for the emotional disturbance is much stronger and more dominant than the intellectual one. The patient quite appreciates the unreasonableness of her foreboding, and

in her mind believes the promises of her physician that her heart is perfectly sound; but there is, despite all this, a distress which destroys her peace of mind and makes her nervous, sleepless, and in every way neurasthenic. Such patients do not have other symptoms of a melancholia, although these types of neurasthenia are sometimes associated, and appear to be almost abortive forms of melancholia. They, however, do not lose flesh; their appetite may remain good, the tongue is not coated, they have no suicidal ideas, nor do they have the persistent insomnia and the agitation of true melancholia. Neurasthenics with morbid fears of places (agoraphobia, claustrophobia), of dirt (mysophobia), etc., and neurasthenics with the doubting mania, are not true cases of neurasthenia, but are suffering from a psychosis.

Angiopathic Neurasthenia.

There are some cases of neurasthenia in which the vasomotor symptoms are extremely prominent. I do not refer now to Basedow's disease, which represents perhaps in a typical way a vasomotor neurasthenia, but to certain cases in which the innervation of the blood-vessels seems to be especially impaired. I have given histories of six cases of a type of this disorder, under the head of "angiopathic neurasthenia."

The patient has the general symptoms of neurasthenia, but in addition he has the special symptoms which consist of a sense of pulsation or beating, which involves the whole body. It is particularly disagreeable, however, on account of the pulsations in the head. The head-throbbing never leaves him, and is so persistent and annoying that it keeps him from work and renders him melancholy and hypochondriacal. The tension of the pulse is low, the rate normal or slightly accelerated. He does not have palpitations of the heart, as in Basedow's disease, and there is no particular dyspnœa on exertion. He has no goitre or exophthalmus. The skin usually shows a striking degree of dermography, and there is an epigastric pulsation, as well as pulsation of the carotid. These patients are nearly all men between the ages of twenty-five and forty. Occasionally a condition resembling this is seen in women at the climacteric. The abuse of alcohol and tobacco, particularly the former, is the conspicuous exciting cause in most of the cases.

Neurasthenia Gravis.

In instances which are fortunately very rare, neurasthenia assumes a very severe and serious type of exhaustion. The patients suffer from the typical symptoms in much the ordinary way, but the

degree of weakness is very much exaggerated. Such patients have not only headaches and disturbed sleep, pains in the back and paræsthesiæ, digestive disturbances, and mental depression, but they speedily emaciate to a considerable extent. They take food in fair amounts, but it gives them no strength. The most careful applications of the "rest cure" secure for them only temporary benefit. They cannot walk far without intense fatigue and exhaustion, with subsequently severe headaches, or even attacks of vomiting and diarrhœa. Despite closest examination, no distinct signs of organic disease can be discovered, and I have known such patients to go on into a permanent and hopeless invalidism which has lasted for many years. In these cases there is not a hysterical or even large hypochondriacal element. No amount of suggestion or "mind cure" has much effect upon them. They are not, in other words, hysterical, bed-ridden women, but often men who have reached or passed the middle period of life, and the condition is one suggesting a premature senescence of the nervous tissues.

Pathogeny and Pathology.

Victims of neurasthenia are persons who in all cases have either inherited or acquired a nervous system with lessened power of resistance. In the vast majority of cases I believe that inheritance is the cause of this weak nerve structure. Such inheritance may be very slight, and, if the patient lives with reasonable care, he has good health and lives to an old age. Under the influence of severe and depressing agencies, or of poisons or infections, however, this resisting power of the nerve cells is weakened. The person then is ripe for an attack of nervous exhaustion.

It seems probable that an inherited tuberculous taint in a measure prepares the system for nervous prostration. Among acquired diseases syphilis undoubtedly impairs the physical strength and makes the person predisposed to neurasthenia. So, I believe, does excessive indulgence in alcohol, tea, and tobacco, and I would add an extreme indulgence in the carbohydrates, such as candies, sweets, and pastries of all kinds, when taken continuously in excess of a normal ratio.

Dr. C. F. Hodge has shown that when the nerve cells are fatigued by persistent work or electrical stimulation, the nucleus of the cells decreases in size, has a jagged, irregular outline, loses its open and reticulated appearance, and takes a darker stain—that the cell protoplasm shrinks slightly in size and stains more feebly. It is a fair inference that human beings who continually and for a long time fatigue their nervous system finally get their cells into a like state and

so disorganize them that they are no longer repaired properly; cell bodies and nuclei become permanently shrunken and lose their normal anatomical structure. This view explains certain forms of neurasthenia that come on gradually as the result of persistent overwork or abuse of the nervous system, with bad feeding and stimulation.

A considerable number of cases, however, including most of the traumatic forms of neurasthenia, come on suddenly as the result of a single severe shock. Here we must invoke some other agency, and this I take it to be the vascular system. Under the influence of intense and sudden emotions of the depressing kind, the vasomotor centre and the whole vascular mechanism go through a kind of convulsion, and this convulsive disturbance is a thing which the vasomotor system of those predisposed to neurasthenia is unable to withstand. In fact, neither education nor past experience has trained the vasomotor system of the present day to bear such profound emotions as are produced, for example, by the experiences of a terrific accident and intense fright. We are no longer a nation of fighters, and are not, as our forefathers were, subject any day to surprises and alarms, but under most circumstances live a life in which sudden and powerful emotions do not enter. A convulsive disturbance of the vasomotor system, therefore, seems permanently, or at least for a long time, to disturb its equilibrium. The nerve cells connected with it are so weakened in their nutritive and functional power that the blood is not carried regularly and normally to the nerve centres in accordance with the way that such centres have been accustomed; hence the nerve cells become impaired in nutrition and functionating power.

Another factor undoubtedly exists in the production of neurasthenia, and that is the irritation of the nerve centres by poisons generated within the body. Mosso, while experimenting upon soldiers who had suffered from the fatigue of a day's march, discovered that the blood of a fatigued animal injected into an animal at rest caused in that animal the symptoms of fatigue. We know that in certain forms of digestive disorder poisons are probably absorbed into the blood, and we know also that in gouty and lithæmic states the uric acid and other products of defective metabolism poison the system and induce many of the symptoms of neurasthenia. There is, therefore, this element of autotoxæmia which probably enters measurably into the production of neurasthenia. The subject, however, has yet to be worked out into definite shape. When a person has suffered from neurasthenia for a considerable time, there are, no doubt, certain more or less permanent changes in the body, at least we note that catarrhal conditions of the stomach and bowels may

become permanent, and that anæmia may be present. In cases occurring in persons advanced in life, arterial changes probably become more rapidly pronounced than in healthy persons. In fact, a prolonged neurasthenia, with the accompanying worry and mental depression, no doubt hastens and accentuates degenerative vascular changes. Dercum has suggested the name "terminal neurasthenia" for that condition of chronic nerve exhaustion in which anatomical changes have become fixed.

Diagnosis.

Neurasthenia is to be differentiated from the following conditions: hysteria, major and minor; hypochondriasis; melancholia; the beginning stage of general paresis; simulation; the reflex effects of some gross bodily disease.

Hysteria major is distinguished by the presence of the stigmata of that condition, and the periodical crises which occur. In the ordinary or minor forms of hysteria the patient does not suffer from any of the classical symptoms of neurasthenia; she often sleeps well, has no persistent headaches, has a good appetite, and has none of the characteristic paræsthesiæ and cephalic sensations; she is mentally active and alert, and often gay and cheerful, and is physically strong. The neurasthenic, on the other hand, is generally depressed and serious and greatly concerned in regard to her condition. She is docile and quite willing to do everything possible to get well. She has no severe emotional crises, and none of the globus or the clavus pains. Hysteria minor may be associated with neurasthenia, and in women this is not infrequently the case. The French have for this combination the term "hystero-neurasthenia."

In hypochondriasis the patient suffers from a purely mental malady. There is almost always a history of hereditary taint, and the patient himself usually shows somatic signs of degeneration. He has few of the stigmata of neurasthenia, and is mainly occupied with a fixed idea concerning some special bodily ailment. It is this isolation of mental symptoms, the hereditary taint, and this very marked evidence of the purely psychic disturbance which sets off hypochondria, as at present understood, from a neurasthenia. Hypochondriacs, it may be added, are bodily well or at least are able to undertake physical exertions, which neurasthenics cannot do. Here, again, however, it must be borne in mind that a person starting with neurasthenia may finally end up with a form of hypochondriasis; that is to say, he gets practically cured of the asthenic symptoms, but his mind has become disturbed by his painful experience, and he settles down into a mild grade of hypochondriasis.

The early stages of mild forms of melancholia simulate neurasthenia. This is so much the case, that some authors have described neurasthenia as an abortive form of melancholia. We have already referred to this under the head of "neurasthenia with fixed ideas," and we there pointed out some of the distinguishing points which enable one to recognize melancholia. The loss of flesh, persistent loss of sleep, rapid pulse, motor restlessness, and extreme mental depression, with delusions and suicidal ideas, are the signs which enable one with very little difficulty to distinguish melancholia. This latter disease, also, is found to simulate neurasthenia mainly in women who are approaching the climateric.

In the early stages of general paresis the patients suffer from neurasthenic symptoms. They find that they are no longer able to work as they did before, their sleep is disturbed, they are excited, forgetful, and nervous. They have not yet developed many of the physical symptoms perhaps, hence their condition suggests and is often mistaken for a simple nervous breakdown. The condition is much more perfectly simulated when the patient has been taking a good deal of stimulation, in order to keep himself up to the mark. A careful examination, however, soon reveals the true nature of the trouble. Even in the early stages of paresis some evidence of failure of memory and of the power to write and spell correctly, with expansiveness of ideas will be found. Besides this, a physical examination will show extreme tremor of the hands, tremor of the face and tongue, and exaggerated reflexes. The pupils also will often be found to be unequal. It is true that facial tremor, tongue tremor, and unequal pupils occur in neurasthenia, but they are rare and not so marked.

A patient may be suffering from a number of bodily ailments, and if this person be at the same time of a somewhat nervous constitution, the condition may resemble neurasthenia. Those persons having a very feeble digestion, with dilated stomach and an atonic condition of the alimentary tract, may get depressed, fretful, and sleepless; so a person suffering from some chronic uterine or ovarian or bladder trouble may present many symptoms of nervous irritation. It must depend largely upon the good sense of the physician to measure the importance of the local troubles as compared with those of the general symptoms. I believe that the fully developed type of neurasthenia is rarely brought out by local disease alone. Still, I have seen cases with neurasthenic symptoms cured for a time by washing out the stomach, and enormous relief to the nervous irritation result from treating the condition of the blood or relieving the uterine disturbances.

Course and Prognosis.

There is such a thing as acute neurasthenia. This follows prolonged debauches and long periods of excessive mental strain, with loss of sleep. Such patients may present all the signs of neurasthenia, and get perfectly well in two or three weeks. Neurasthenia, however, is essentially a chronic disease, and when speaking of it, we refer to this type of the disorder. It is a disease which comes on as a rule gradually, developing, however, in the course of a few months. It may come on suddenly after shocks and accidents, and it may develop or follow rapidly after an acute infectious fever. It reaches its height in a comparatively short time, and runs a course lasting from one or two to seven or eight years. Its course is a varying one, and this variation is particularly noticeable when the patient begins to get well. The patient continues to improve for a time and then suddenly falls back, then goes forwards again and thus convalescence progresses. Complete restoration to health is possible and frequent, but the patient always has to take more care of himself than before. As a result of an attack of neurasthenia, men and women who have suffered from it are apt thereafter to lead very saint-like and ascetic lives, and hence they as a rule live long. It used to be said by Dr. Beard that neurasthenics would have a long and happy old age. They pass through the valley of the shadow of death, but the experience may be a profitable, if not a pleasant one.

Treatment.

Naturally, the measure of leading importance in the treatment of neurasthenia is rest, and the problem of how this can be obtained is the first one to confront the physician.

In the severe types of hystero-neurasthenia, especially when it occurs in young women, the application of the "rest cure" which has been so ingeniously elaborated and perfected by Dr. Weir Mitchell, is undoubtedly the best treatment. I do not find, however, that men submit themselves readily to this measure, and it seems to me to answer best in those neurasthenic women who suffer also from some hysteria, and who are reasonably "suggestable" patients. A modified rest cure can often be secured by making the patient stay in bed until after midday lunch or lie down for an hour after each meal, and go to bed early in the evening. Business men will often cut their business hours down one-half if they are allowed still to continue some work. I do not believe, however, that the physician should often use half-way measures, and it is best to impress at

once upon the patients the fact that nothing is of so much importance to them as to get well, and to get so that they can take their place at their work again. Change of scene is usually very beneficial to neurasthenics, but travelling is injurious to them. They should be sent to some special place and be made to stay there. A tour along the Mediterranean coast or a trip to Europe often brings them back worse than when they went. Much the same can be said of trips to various places in the South or West. Some of the sanitaria in Germany, some of the places in the Riviera, Egypt and Bermuda, parts of North and South Carolina and Arizona, furnish good resorts for neurasthenics. They generally do better in the mountains, if the altitude is not too high, than they do by the seashore. Dry, windy, sunny climates like the Colorado plateau and parts of California and the Northwestern States are too stimulating for most cases.

Much good may be obtained at the numerous sanatoria which exist in this country. Many of these are well conducted and well supplied with all the modern appliances for treatment. It is, however, always a serious thing to send a neurasthenic to a sanatorium, for the reason that if he stays there too long he becomes contaminated with the atmosphere of invalidism about these places and develops hypochondriacal ideas as to his diet, his liver, his stomach, his sleeplessness, and his various sensory disturbances. In sending a patient to a sanatorium, it is a wise plan to tell him not to stay, under any consideration, longer than six weeks; usually four is better. In the summer time great benefit can be secured by camping out in the woods and living a purely outdoor life, away from all the conventionalities and restraints of civilization.

The diet of neurasthenics, according to the views of most American physicians, should be chiefly a nitrogenous one, and my directions are that the patient can eat meats, fish, eggs, green vegetables, and fruits. Milk can almost always be taken, at least for a short time. There is a certain class of lithæmic patients who do best upon milk, vegetables, and fruit, with practically no meat; these, however, are in the minority. In general, tea and coffee, alcohol and tobacco, are to be entirely prohibited, but this is not an absolute rule. In some cases coffee is beneficial, in some tea does no harm, and in others a small amount of whiskey or dry wine and a cigar are also harmless. The physician has to determine this by the reactions and habits of the patient. Neurasthenics usually drink too little water and it is wise to prescribe a certain amount for them. Four or five glasses of water, which may be either plain or alkalinized, are to be taken daily. I find no special advantage in the various

much advertised lithia and spring waters. In dyspeptic patients the meals should be small in amount and taken at frequent intervals; three light regular meals a day and a little food in between form a regimen which usually answers well.

Hydrotherapy, electricity, and massage are all measures which prove of service to the neurasthenic. Of these, hydrotherapy is the most useful, though its value can be overestimated. The ordinary prescriptions consist in the cold sponge bath every morning, and, if it is practicable, the use of a Charcot or a Scottish douche every other day. In women wet-packs with massage are sometimes helpful, particularly in cases in which there is a great deal of nervousness and motor irritation. At night a lukewarm bath, at a temperature of 95°, for ten minutes sometimes relieves the paræsthesia and sleeplessness.

Massage seems to me of not very much use in men, but it is often grateful and helpful to women, and when a great deal of rest is to be enforced, it is essential to employ it for both sexes.

Physical exercise is an agent of enormous value in neurasthenia, and the advent of the bicycle has done a vast deal of good in relieving this condition. Horseback-riding is probably just as efficient, but much less practical. Many persons are greatly wedded to the exercise of walking, and it seems to best fit their needs. It is, however, a kind of exercise which does not take the patient's mind off himself, and does not develop the respiratory functions as well as other measures do. Golfing fills in this lack, and this sport will doubtless be of service in neurasthenia.

The drugs of most value are the bromides, nux vomica, mineral acids, quinine, iron, valerian, the coal-tar antineuralgics, the hypnotics, and saline and alkaline laxatives and salicylates.

The bromide of sodium or potassium should be given in small doses; it should be kept up for a limited time and then gradually reduced. At the same time or later the patient may be given a tonic mixture containing such drugs as the symptoms suggest. Quinine must be given carefully, as it causes increase of nervousness in many.

Phosphoric and muriatic acids are the two mineral acids most often of use. These acids are usually better given after meals. The saccharated carbonate of iron or Blaud's pills, if given, should be given generously, *i.e.*, in doses of thirty grains daily. The best preparations of iron are the tartrate of iron and potassium, the carbonate, the citrate, and the tincture. I find no especial benefit from the albuminate or the peptonized preparations.

The foregoing covers in a general way the measures to be used in

treating neurasthenics. The physician must seek to secure the complete confidence and docility of his patient. He then uses measures which secure some bodily and much mental rest. He gives to him simple and nourishing food in no excess, and prescribes measures which restore the slowed-up or perverted metabolism.

THE DISORDERS OF SPEECH.

BY

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DENVER.

THE DISORDERS OF SPEECH.

By speech is to be understood the sum-total of those processes by which ideas are received from or communicated to another mind. The disorders of speech are not only of the greatest interest as psychological phenomena, but in practical medicine, as symptoms of localized brain disease, they are second in importance only to the paralyses.

When the attempt was first made, some thirty-five years ago, to localize the faculty of speech in the brain, but one centre for speech was spoken of, that now known as Broca's convolution or the motor speech centre. It was soon found, however, that disease of other parts of the brain interfered with the use of language, and we now know that there are four cortical centres necessary for the full use of words: the auditory centre by which words are heard, the motor speech centre by which they are uttered, the visual centre by which they are seen, and the writing centre by which the motions of the right hand are coördinated in writing. These four centres are connected one with another and also with their corresponding peripheral organs, the ear, the vocal apparatus, the eye, and the muscles moving the hand, and with those parts of the brain concerned in ideation, that is, with all the cortical centres for sensation.

Hence it is evident that speech is a very complicated process. It includes the action of the eye and ear, the flow of nervous impulses from these organs to the auditory and visual centres in the cortex, the play of association impulses between these and other sensory centres arousing ideas, and, finally, the excitation of the peripheral organs to utterance or writing. It is impossible to understand or remember the different ways in which speech is disordered without a definite conception of the relation of these processes to each other. This can best be gained by studying the orderly development of language processes in a child, considering each step in the light of our present knowledge of the localization of functions in the brain. It is a great merit of Wernicke and Lichtheim that they have tried to represent accurately, by means of diagrams, the action of the brain centres in the different ways of using language and the defects that must result from disease of the various centres or conducting paths. Some objection has of late been made to their methods, but with-

out good reason. If we think of these processes as physical
we must have the aid of mental if not of actual diagrams, and
gram actually drawn makes our thought sufficiently precis
rigorously tested by comparison with the facts of clinical obse

The Acquisition of Language by a Child.

The Meaning of Words.—The words which a child hears
the first months of its life are to it sounds without significanc
first step it takes in the acquisition of language is to learn th
ing of a few of them. The nerve currents started in the inte
travel along the fibres of the auditory nerve to the auditory
in the medulla, whence fresh impulses are sent to the auditory
in the cortex. It is now quite definitely established that the a
centre of each hemisphere is the posterior half or two-third
first temporal convolution and the adjacent part of the second
only when impulses started in the ears reach these centres an
them to a discharge of nervous energy that the sensation of s
curs. Each ear is connected mainly with the cortical centre
opposite side, but also to a considerable extent with that on t
side. As a mere sound, a word is probably heard equally
the right and left auditory centres, but it is only through the
that this sound has a meaning.

Now, the meaning of a word is always the memory of a se
or group of sensations. The meaning of the word red, for e
is the memory of the color red. It is one of the fundamental
psychology that if two sensations frequently occur at the sam
or in close succession, one of them occurring afterwards alone
call the memory of the other. After a child has seen the color
at the same time heard its name, the two sensations become ass
what was a mere sound now revives the memory of a color an
a meaning. How can we picture this association as a physic
ess in the brain? Each time that the sensation red occurs th
cal visual centre (V, Fig. 37) is excited in a peculiar way and i
such a condition that it will be more easily excited in the sa
in the future. Moreover, as the nerve cells in this region
nected with other parts of the brain by nerve fibres, the ex
is not limited to them alone, but tends to arouse other centres,
these the auditory centre, A. If A is excited at the same tim
sound of the word red, it also tends to excite other centres
its connecting fibres, so there is a double excitation along t
AV. The oftener this double process is repeated the less the re
along the path AV and the more ready each centre is to res

an impulse from the other. Finally the two centres become so related to each other that whenever the sound of the word is heard the auditory centre sends to the visual centre an impulse strong enough to excite it in the same way, although not so vigorously, as the actual color would. Instead of the color sensation we have the memory or idea of it, which is said to be associated with the sound of the word. The fibres connecting the two centres are called association fibres. Two associated ideas, however, do not necessarily exist in different

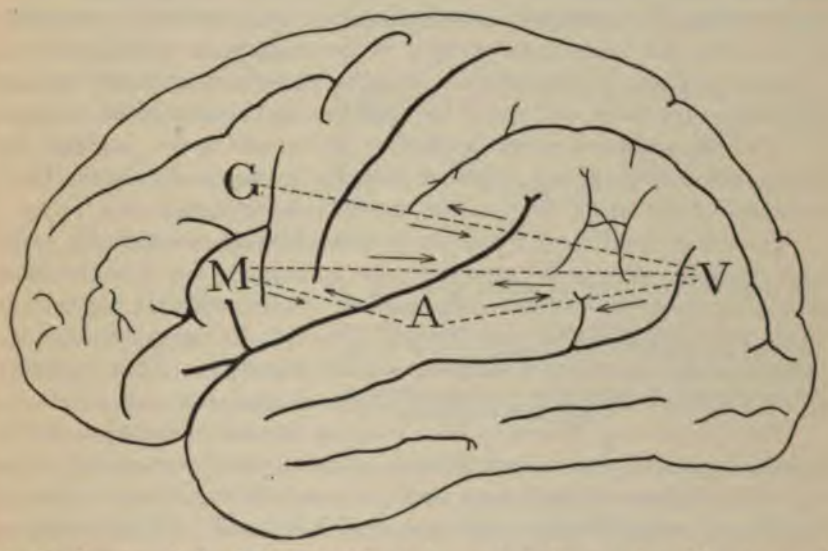

FIG. 37.—Diagram of the Left Cerebral Hemisphere. *A*, Auditory centre; *M*, motor-speech centre; *V*, visual centre; *G*, centre for coördination of the movements of the right hand in writing. The dotted lines indicate association fibres and the arrows the direction of association impulses.

centres; both are often in the same centre, in which case the association is said to be intracentral.

By a precisely similar process the memory of the sensations sweet, bitter, sour, and salt has become associated with the sounds of the corresponding words; but in these cases the association impulses from the auditory centre, instead of passing backwards to the centre for visual memories, pass forwards and inwards to the centre for taste. In the same way such words as stench and perfume have their meaning in the centre for smell, smooth and rough in that for touch, heavy and light in that for muscular sense, sting and ache in that for pain, and so on. It will be readily seen that words may be divided into classes according to the sensory centre that is aroused and the kind of sensation that is recalled when the word is heard, and that, as

these centres are in different parts of the cortex, the associati for one class of words must be quite distinct from that for a The practical importance of this lies in the fact that a lesion so placed as to interfere with the association impulses going t one centre, thus preventing words that appeal to that centr being understood, while words of other classes are readily c hended.

Most words, however, do not have for their meaning a singl memory, but a combination of such memories. The sound word orange, for instance, arouses in the visual centre the n of the color and form of an orange; in the centres for touch an cular sense ideas of smoothness, roundness, weight, and solid the centres for taste and smell the tartness and aroma of an All of these scattered sense memories are bound to one anot association impulses and, together with the memory of the sou utterance of the word, they constitute the concept orange.

The understanding of words is, of course, interfered with b nary deafness, simply because they are not heard; but it is al that disease in or near the left auditory centre, without pre words from being heard, may prevent them from being underst cutting off the association impulses to other centres. This in to understand words that are heard is called word-deafness.

The Utterance of Words.—After learning to understand a few a child begins to utter some of them. The simpler movement vocal organs have already been acquired, and their cortical cen the lowest part of the motor area are well developed. But to c these movements so as to form articulate words another centre into action, the motor-speech centre or Broca's centre, situated base of the left third frontal convolution (M, Fig. 37). In stored the memories of the movements necessary for the artic of each word.

To combine the elementary sounds into words this centre of course, control the action of the lower nerve centres for th organs, that is, the motor nuclei of the fifth, seventh, tenth, el and twelfth nerves. Whether this control is effected through rate path leading directly to these nuclei, or by regulating the of the cortical centres for the simpler movements, which in tu trol the nuclei, is not yet known. It is known, however, tl fibres conducting speech impulses to the nuclei pass through ternal capsule where they frequently suffer injury.

The first utterances are imitations of words frequently he the child, probably without any sense of their meaning. This tive process is an acquired reflex. The auditory centre, on per

the sound of the word, sends impulses to the motor-speech centre, arousing it to send such impulses to the vocal organs as will cause the word to be uttered. It is important to note that the motor processes of speech develop only under the guidance of the auditory centre. Deafness occurring in childhood not only prevents further progress in learning to talk, but, up to the age of four years, and in some cases much later, it causes the loss of the power of articulation already acquired. On the other hand, the motor-speech centre has a very important influence upon the auditory centre. The sound of a word is much more distinct when we remember its utterance, no doubt owing to association impulses passing backwards from Broca's centre to the auditory centre.

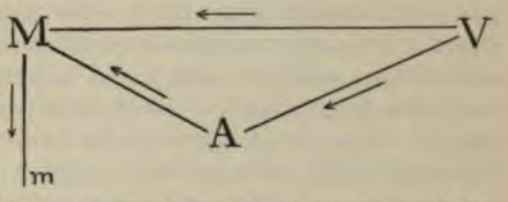

FIG. 38.—Diagram Illustrating the Cerebral Process in Uttering a Word Corresponding to a visual Idea, or in Reading Aloud without Understanding what is Read. *V*, *A*, and *M*, visual, auditory, and motor-speech centres. The association impulses, starting from *V*, flow as indicated by the arrows, and end in the impulses *Mm*, going to the motor nuclei of the vocal organs.

Although the first utterances are imitative reflexes, a child soon begins to use words to express its meaning. It is evident that the process of expressing ideas in words must begin in the centres for ideas, that is, in the sensory centres. How do these centres act upon the motor centres so as to cause the utterance of the appropriate words? Let us suppose that the idea of redness occurs in the mind of a child, and that as a result it utters the word red. The idea is a visual memory resulting from the activity of the visual centre (V, Fig. 37). We might expect this centre to excite the motor-speech centre directly, by the path VM, and no doubt it does; but this direct excitation is an inefficient one. If the auditory centre is destroyed, the rest of the brain remaining intact, the patient is able to talk, but only in an unintelligible jargon. In such a patient the idea of redness, instead of causing the word red to be pronounced, would call forth a different word, or perhaps an unintelligible sound. This shows conclusively that the coöperation of the auditory centre is necessary to the correct expression of ideas. We must suppose that the motor centre works under the influence of two sets of impulses, as shown diagrammatically in Fig. 38. In the case supposed one impulse, VM, arouses the motor centre, but does not determine what it shall do. At the same time another impulse, VA, arouses in the auditory centre the memory of the sound of the word, and this centre then sends an impulse, AM, to the motor centre insuring the utterance of

the correct sound, just as it did when words were first utterec tative reflexes.

What has been said of the idea of redness and its expres obviously apply just as well to other visual ideas, and also tc ideas occurring in other sensory centres. But most words ideas which are not simple but consist of two or more sense n having their seat in separate regions of the cortex. On ex such an idea it will be found that one of its component sense n predominates over the others, and that this is usually the vis Think of any object and its visual image at once appears in co ness. Even molecules and atoms, which no one has ever s pictured to ourselves as visible. So prominent is the visual i the idea of most objects that it might seem to be the entire idea closer examination we readily see that with the memory of t and color of an object are associated sensations of touch and n effort. When we picture feathers to ourselves we feel that t light and fluffy, and when we imagine a cannon ball we feel tl solid and heavy. The predominance of the visual component ideas makes it extremely probable that in spontaneous speech t cipal association tracts by which ideas recall the sound and utte the proper words are those from the visual centre to the audit motor-speech centres. At the same time all the sensory ce which the qualities of an object are remembered probably ai calling the name, and impulses from any of them may alone cient. Thus the name of an apple may be recalled by seein distance, or by feeling, smelling, or tasting it with the eyes Writers on this subject, while fully recognizing the fact that no single centre for ideas, have, for convenience, represented centre in diagrams of the speech processes. There can be n tion to this if it is always remembered that this diagrammati for ideas stands for any or all of the sensory centres; otherw likely to mislead.

Broadbent, followed by Mills and others, has supposed special concept or naming centre exists in the sensory par cortex, and that in the motor part there is a special "propositic centre whose work it is to arrange words into sentences. N standing the great consideration due the individual suppo this hypothesis, it has generally been rejected as superfluou cent writers.

As a child's command over language increases, there is sponding growth in intelligence, and that this growth is lar pendent upon the ability to receive and express ideas by n words is shown by the slow development of intelligence i

mutes. How far does thought depend on words? is not only an interesting but a very practical question, for it is often important to ascertain the mental condition of a patient whom disease has deprived of the use of language. That it is possible to carry on very definite and coherent mental processes without the aid of words must be evident to those with any power of introspection whatever. One familiar with football can recall every incident of a game as it would appear to him without necessarily thinking of a single word. The players in such a game, in deciding where to throw the ball or how to elude opponents, make exceedingly rapid inferences of a highly rational character without the use of words. The chess-player contemplating a move may foresee in a series of visual images, without accompanying words, many of its possible consequences, and govern his action accordingly. Now, to most people, such mental processes are thought, and therefore they say that thought without words is not only possible, but constitutes a large part of subjective experience. This is in accord with the fact that aphasic patients often play games and even transact business with considerable intelligence.

On the other hand, it is equally plain that most of our thinking is done by means of words. The examples of thought that have been given include concrete ideas only. Abstract ideas require the aid of words to bring them into consciousness. We may think of red, blue, or yellow without the corresponding words, but we can have no idea of color in general without a word, or at least a sign, for it. The more abstract an idea is the vaguer are the sense memories constituting it. This must be so because an abstract word stands for any one of a number of different concrete ideas, as an algebraic letter represents any one of different quantities. The word is the only sense memory that is associated with all the concrete ideas. Take it away and the individual ideas remain, but there is nothing to unite them into one group; the general idea has disappeared.

For all inferences drawn from general truths, for the careful weighing of evidence, for the deliberate consideration of the consequences of any but the simplest action, abstract ideas are necessary and so are abstract words. It should be said, however, that there is some very strong testimony contradicting this statement. The French professor of physiology, Lordat, who for a time suffered from a complete loss of the memory of words and afterwards recovered and wrote an able analysis of speech, said that while the loss of words was complete, he could think perfectly well. This testimony has generally been disbelieved, yet it certainly seems arbitrary to disregard a positive statement from so competent a witness without at least some explanation of how he could be so mistaken in re-

gard to his own mental processes. It seems to me that explanation can be furnished.

Theoretically abstract words represent equally all the qualities or objects generalized by them, but this is not stri The word color will habitually arouse the idea red in on blue in another, and green in a third, although it is fully u that it applies to all colors. The word beauty to painte ideas, more or less definite, of agreeable forms and colors, cians memories of delightful sounds. Lordat may have l associated vivid concrete ideas with abstract words, and th alone, revived in proper order, may afterwards have been for abstract ideas. He claimed, for example, that he fully ated the meaning of the formula, "Glory be to the Father a Son and to the Holy Ghost," without being able to recall word of it. If we suppose that with the word glory he had l associated the idea of a bright light or a burst of music, wi the visual image of a venerable and majestic man, with Sor ventual image of Christ, and with Holy Ghost the image o we can readily understand how these sensory images might l in order during a religious service, and seem to be the full of the absent words, which, of course, they were not. In way, when he thought of the subject matter of his lectures jected improvement in their arrangement without any recoll the words, it is probable that what he really considered was l representation of physiological processes and the sensations ally associated with the delivery of the lectures.

Without dwelling longer on this interesting question, we clude that aphasics who cannot recall the sound of word those who have learned to substitute signs for words) cann any course of thought involving the use of abstract ideas.

The power of spontaneous speech may be lost in a variety Speech will, of course, be prevented by destruction of Broca or of the tracts connecting it with the bulbar nuclei for spe these nuclei themselves. It is also greatly impaired by dise auditory centre, so that paraphasia (the substitution of wro and elementary sounds for the right ones) is, next to word- the most important symptom of auditory aphasia. Moreove of any of the centres for ideation, or of the tracts connecti the auditory and motor-speech centres, interferes with the sp utterance of any word whose idea resides in this centre. ease of the visual centre (angular gyrus) or of the region jus of it interferes with the naming of colors, forms, and ordinar

Reading.—When a child begins to learn to read it has

large store of words which it readily understands and utters. Learning to read consists essentially in the arbitrary association of elementary sounds with printed letters, and in learning, at the sight of a word, to utter in quick succession the sounds represented by the letters. At first each letter must be seen separately in order that its sound may be produced, but after practice the appearance of a familiar word as a whole recalls its utterance as a whole without the intermediate process of spelling. The mere production of the sounds at first so occupies the attention of the beginner that their meaning is scarcely considered. But the learner very soon perceives that the sounds, thus mechanically produced, are familiar, and he becomes conscious of the meaning of what he reads, just as if it were spoken to him. For a time it is necessary to read aloud in order to understand; the printed characters do not represent ideas, but sounds, which have previously become associated with ideas, and they have no meaning until they are translated into the corresponding sounds.

With practice, however, it becomes possible to suppress the actual utterance and still understand what is read; even the slight movements of the vocal organs, which persist so long in children, are ultimately dispensed with. Does this mean that the nature of the process has changed, and that ideas are now directly associated with the printed words? No, the reader is still conscious of every movement of the vocal organs that he would make in uttering the words and of the impression that every word would make upon his ear. In memory he both feels and hears himself talk.

This much may be deduced by each one from his own subjective experience. Now let us consider the physical process of reading as it goes on within the brain. Evidently this begins in the half-vision centres with the reception of visual impulses from the retinæ. Here the sensation of sight occurs, but it is the general belief that visual memory has a separate seat in the angular gyri, and that the memory of printed or written words resides only in the left angular gyrus.

It seems to me highly improbable, notwithstanding the eminence of many who hold this opinion, that the memory of a sensation should be in any other centre than the one in which the sensation originally occurred. Yet it is a well-established fact that damage to the left angular gyrus prevents written or printed words from being understood, and impairs or destroys the memory of their appearance, and this is taken as proof that the angular gyrus is a higher visual centre, especially devoted to the preservation of visual memories, while those cortical areas about the calcarine fissure on each side (the half-vision centres) are concerned only in the sensations of sight. I believe the

true explanation of the fact is the one advanced by Lissauer, substantially as follows:

The projection, through connecting nerve fibres, of the image upon the half-vision centres suffices for sensations of co only very imperfectly for those of form. Our finer perception depends upon the movements of the eyeballs in following the of an object. The angular gyri are, strictly speaking, not visual centres, but centres for the supplementary sensations, associated with those of vision, caused by the movements of balls. If these sensations of movement do not occur the finer tion of form is lost, and if they cannot be recalled the me form is lost. As the movements of the eyeballs are so clo sociated with the strictly visual sensations in the half-vision each angular gyrus is directly connected with the adjacent ha centre, and, as the eyes must move together, the two angular directly connected with each other by fibres of the corpus ca The forms of ordinary objects, and perhaps of figures, are p and remembered by either angular gyrus, but the more difficu of letters and words are accurately perceived and remembe by the left one. With this explanation (which accords w Ferrier's early experiments) the left angular gyrus will be co as the visual centre, so far as the use of language is concerne

When a word is seen its form is perceived by this centre, sociation impulses (VM, Fig. 38) arouse the motor-speech c action; at the same time other impulses (VA) go to the a centre, recalling the sound of the word. The auditory cent sends impulses to the motor centre, insuring the utterance (b of the impulses, Mm, to the vocal organs) of the correct sound this guiding impulse from the auditory centre is necessary cases is shown by the fact that when the auditory centre is de reading aloud is generally prevented altogether, or the wrong are uttered, just as spontaneous speech is perverted in such In this respect, however, all cases are not alike. In two c ported by Starr, the patients could read aloud correctly, understanding, although the auditory centre was presuma stroyed. In these cases the impulses VM must have bee ficient guide for the motor-speech centre without the coöper the impulses AM.

As each word is uttered it is heard and understood by th just as if spoken by some one else, that is, by means of impul Fig. 39) from the ear to the auditory centre and association i (AI) from it to the sensory centres, where the idea correspo the word is aroused. At first actual utterance is necessary i

to stimulate the auditory centre to send sufficiently vigorous association impulses to the centres for ideas. Later, when practice has made the whole process easier, a vivid memory of the sound of the word in the auditory centre is enough to cause the association impulses to be sent out. The impulses Mm and aA are then dispensed with. Nevertheless the memory of the utterance remains in the motor-speech centre, and it plays a very important part in quiet reading, as is shown by the well-known fact that in most, if not in all, cases of destruction of the motor-speech centre the understanding of what is read is lost. The explanation of this curious fact is that the memory of the utterance of a word, by a reflex action on the auditory centre through the impulses MA, makes the memory of its sound particularly vigorous, and thus causes the association impulses reviving the idea to be stronger than they would otherwise be. The utterance memory is still needed to reinforce the auditory centre just as actual utterance was necessary at first.

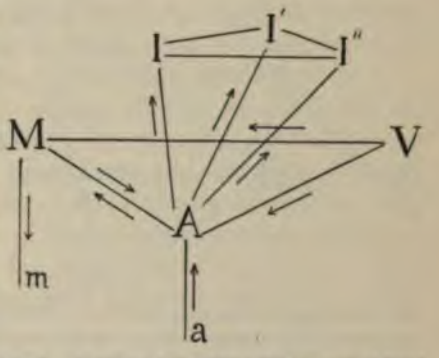

FIG. 39.—Diagram Illustrating the Process of Reading Aloud and Understanding what is Read. *V*, *A*, *M*, and *Mm*, as in Fig. 38; *aA*, tract by which impulses from the ears reach the auditory centre and enable the reader to hear what he reads; *AI*, *AI'*, etc., association impulses from the auditory centre to the various centres for ideas. In quiet reading the tracts *Mm* and *aA* are not used, but the auditory centre is reinforced by the reflex impulses *MA*.

That this explanation of the cerebral process in reading does not apply to the reading of deaf-mutes is obvious. Such patients, having no store of significant sounds which the printed words have merely to recall to mind, must associate ideas with the visual words, either directly or by the intermediation of gestures, a much more difficult process. No doubt normal individuals could do the same thing, but they do not, because, the number of elementary sounds being very limited, it is much easier to associate each printed word with its sound than with its meaning.

It is evident that there are many positions in which disease may interfere with the cerebral process of reading and thus cause alexia, the inability to understand written or printed language. Disease in the region of the left angular gyrus does not prevent words from being seen as visual objects, but by interfering with the accurate perception of their form, or with the sending of the association impulses to the auditory and motor centres, it prevents both the sounds and the ideas from

being recalled. This condition is known as word-blindness
alexia. If the auditory centre be destroyed, the visual and
centres being intact, words may be uttered in reading; but
they are the wrong ones and are not correctly pronounced,
if reading aloud should be correct the meaning is usually
or altogether lost. The inability to read aloud correctly cor
to the paraphasia which it accompanies, and is called p
The inability to understand is called auditory alexia. W
motor-speech c
destroyed,
aloud is of cou
but it is also a
of common obse
as was early r
by Trousseau,
patient has littl
comprehension
although he ma
to occupy hims
books and paper

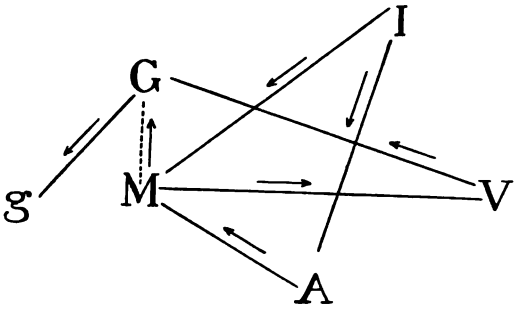

FIG. 40.—Diagram Illustrating the Process of Writing. Centres for ideas, I (represented as single for the sake of simplicity), send impulses to *A* and to *M*, reviving memory of sound and utterance; *A* sends impulses to *M*, insuring correct memory of utterance; *M* sends impulses to *V*, recalling the appearance of the words, and *V* to *G*, recalling memory of the hand movements; *Gg*, the outgoing impulses towards the muscles moving the hand; *MG*, short cut for words written automatically when *V* and its connecting tracts are not used.

Learning to
This consists
copying lett
words without r
to their meani
then in associat
necessary movements of the hand with the memory of the ap
of the word and its appearance with its utterance. The pr
spontaneous writing begins in the sensory centres with the oc
of the ideas to be expressed. From these centres association i
(IA and IM, Fig. 40) go to the auditory and utterance cent
recall the memory of the sound and utterance of the appropriat
Just as in spontaneous speech, impulses (AM) from the aud
the motor-speech centre are necessary to insure the correctne
utterance memory. Actual utterance may or may not occur,
memory of it is absolutely essential. Then association i
(MV) from the motor-speech centre to the visual centre recall
pearance of the written word, and another set of impulses (V
the visual centre to the centre for coördination of the moven
the right hand causes the word to be written. For one's ow
ture the recollection of its appearance does not seem to be ne
the association impulses pass directly from M to G, or even f
G, without the intervention of V. It is also probable that i

individuals who write a great deal many words may be thus automatically written without the recollection of their appearance; still, the process first described is the usual one.

The complicated nature of the process of writing makes it possible for agraphia, the inability to write, to be brought about in many ways. Destruction of the motor-speech centre always causes agraphia because it prevents the formation of the words to be written. We have no short term for this most common form of agraphia. I would suggest motor-speech agraphia as the term best describing it. Disease of the motor-speech tract below Broca's convolution, however, while it causes one form of motor aphasia, does not interfere with writing because the utterance of the words can be remembered.

Disease of the auditory centre affects writing in precisely the same way as it does spontaneous speech, but to a still greater degree. So, corresponding strictly to the paraphasia and paralexia, already mentioned as symptoms of disease of the auditory centre, we have paragraphia, that is, substitution of wrong letters for the right ones in a word and of wrong words for the right ones in a sentence. This may be so extreme that the patient will make no attempt to write; we then have auditory agraphia.

Disease destroying the left angular gyrus or cutting off communication between it and the utterance centre will necessarily prevent the appearance of the word from being recalled when it is uttered, and this will generally prevent its being written. Inability to write from this cause is called sensory agraphia, because due to the absence of the memories of the sensations which normally guide the movements of the hand. Visual agraphia would be a better designation because loss of muscular sense might also cause a sensory agraphia.

Finally, the proper words may be formed to express the ideas, and their appearance may be recalled perfectly, and yet writing with the right hand may be impossible on account of disease of the centre coördinating its movements. This is called motor agraphia, although the incoördination may be really due to a sensory loss. It should be noted that this is not a complete agraphia, for the patient can still make the motions of writing with the left hand or other movable part of the body. The seat of the lesion is probably the foot of the left second frontal convolution. This centre seems to have the same relation to the centres for the simpler movements of the hand that Broca's centre has to those for the simpler movements of the vocal organs.

Relation of the Right and Left Hemispheres in Speech

Each of the speech processes has so far been assumed to ried on only by the left hemisphere, but this is not strictly tr right-handed persons only the left hemisphere is sufficiently to carry on the difficult cerebral processes of speech, but eac on the left side is directly connected with its rudimentary f the right, so that when the left centre is in action the right to coöperate to a limited extent. When left to themselves t centres are nearly helpless, but they are usually capable of work which, in some cases, can be greatly increased by ex the left centres are disabled. In left-handed persons the processes are carried on by the right hemisphere.

Method of Examination in Disorders of Speec

In addition to the thorough examination that should be gi case of serious nervous disease, patients exhibiting a dis speech require a systematic and minute investigation of thei to comprehend and use language. The special symptoms noted according to the following scheme:

Acuity of general and special sensibility.

Comprehension of oral speech.

Imitative speech, ability to repeat words heard.

Associational speech, counting, saying alphabet, days of w

Spontaneous speech.

Naming objects, not only when seen but when perceived b senses than sight.

Selection of an object named from among a number.

Recognition of the nature and use of objects as tested by c senses.

Reading aloud.

Comprehension of print or writing.

Naming numerals, letters, words, etc.

Pointing out numerals, letters, words, etc., when named.

Copying.

Writing from dictation.

Associational writing, numbers, days of week, etc.

Spontaneous writing.

Ability to perform arithmetical operations.

Appreciation of music.

Gesture language.

Mental condition.

In testing the comprehension of speech, commands should be given for the patient to execute, proceeding from the simpler to the more difficult and taking care that no gesture or change in facial expression gives an inkling of the meaning.

Sometimes the comprehension of words appealing to different sense memories should be separately tested, for example, red, loud, perfume, bitter, smooth, warm, cold, heavy, sting, etc.

In testing spontaneous speech it is important to note the choice of words, their arrangement in sentences, and their formation by the vocal organs. The comprehension of print or writing should be tested, not by asking the patient if he understands what he reads, but by seeing whether he can in any way give an accurate idea of it. A good plan is to give written directions and note whether they are accurately fulfilled. Gesture language may sometimes indicate the patient's mental state when words are neither used correctly nor comprehended. It is always important to note the mental condition, because absence of speech may be due to the general depression of melancholia, to dementia or idiocy, or to the influence of a delusion. Before Broca's time an aphasic patient was very likely to be thought insane; now there is more danger of certain insane patients being thought to be aphasic.

Stuttering.

Stuttering (anarthria spasmodica) is a sudden arrest of utterance due to spasm of the lips, tongue, soft palate, glottis or respiratory muscles. This spasm is a prolongation and intensification of the muscular contractions that occur in normal speech, particularly of those which momentarily stop the flow of expired air. The difficulty is not in making any of the elementary sounds but in relaxing the muscles for one sound in time to produce the next.

The spasm is especially apt to occur in the production of the explosive consonants, b, p, d, t, g (hard), and k. It occurs less frequently with the continuous consonants, m, n, v, f, th, z, s, sh, y, and w. Only rarely does it occur in the glottis, on attempting to produce the vowel sounds, and still more rarely in the muscles of respiration, unduly prolonging expiration or inspiration or perhaps causing a disturbing rhythmical catch in the breath. It is more apt to be on a consonant at the beginning than on one at the end of a syllable.

When the arrest occurs on a consonant it may be complete and prolonged until the patient with a great effort overcomes the obstruction and continues, thus: bb----oy; or the spasm may be partly overcome and then renewed, causing a repetition of the consonant, thus: b-b-b--oy. Spasm of other parts of the body, especially of the face,

often accompanies the arrest of speech. In an extreme case is first red and afterwards blue, with swelling of the veins, anc sive movements beginning in the face may spread to the enti This pitiable state continues until impending asphyxia con patient to give up his attempt at speech.

Stuttering is not a constant symptom in any case; even t stutterers are free from it at times. It is most likely to oc the patient feels himself to be under the observation of oth ticularly strangers, and when he fears that he may stutte anxious not to. During unconstrained talk with his famil mate friends it is in comparative abeyance. In solitude and it entirely disappears. Rhythmical and melodic utterances singing and intoning are much less likely to be marred by s than is ordinary speech.

Any disease that irritates the brain may be a cause of st but in the great majority of cases it is a purely functional and it is as such that it will be further considered. Three-f the cases show an inherited predisposition to neurotic dise other physical or mental stigmata of degeneration are often by no means always, present. The patients are commonly sionable, shy, and easily confused. One-third belong to that have more than one stutterer. The onset is generally a almost always occurs in childhood or youth. In nine-tentl cases it is before the tenth year, but it may be at any age.] three times more liable to it than girls. Insufficient develo speech for the age of the child may for a time precede tl onset. The most common exciting cause is fright, whether panying injury or not. Infectious diseases and imitation n precipitate the onset.

The prognosis depends on the degree of inherited taint tensity of the neurotic temperament, and the duration of th There is some tendency to improvement as age advances, n on account of the increased composure of middle life and a age. In general, the outlook for permanent cure is bad, fo under favorable circumstances and skilful treatment tempora may usually be obtained, the probability of relapse is very gr

The general treatment of stuttering is the same as that of thenia attended by morbid fears. Everything that is possible be done to secure tranquillity and serenity of life, with stric ance of overwork and excitement, but with sufficient exerc recreation. The drugs that are most likely to be useful tonics, particularly strychnine, arsenic, and iron. Bromides of considerable service in subduing nervousness on a particul

sion, but their administration should not be prolonged. Cold sponging is valuable on account of its general roborant effect. Any local disease or malformation of the organs of speech should, of course, receive proper treatment. If any organic disease of the nervous system exists its importance will generally overshadow that of the stuttering.

The special treatment consists essentially of speech gymnastics, proceeding gradually from the very easy to the most difficult exercises in such a way as to restore the patient's confidence in his own powers. These exercises can be carried on far better in special institutions than in ordinary practice. In most cases it is best to begin with a period of absolute rest to the vocal organs. The first exercises are in full and rhythmical breathing; all recent writers agree that in stuttering respiration is always more or less at fault. Then the production of vowel sounds, prolonged through an entire expiration, is practised, taking care to secure purity and smoothness of tone. Next single syllables, beginning with a vowel and ending with a consonant, are taken up, and after the consonants in this position are mastered syllables beginning with consonants are tried. If at any time stuttering occurs the patient must instantly stop and, after a short rest, try again. From this an advance is made to polysyllabic words and to sentences, taking care to secure full inspirations at the right place together with smoothness of the vowel sounds and a rhythmical utterance. The patient should finally be very gradually accustomed to converse with strangers.

Stammering, Syllable-Stumbling, and Scanning.

These defects are characterized by difficulty in producing some elementary sounds or certain combinations of them, as a result of which the sounds are omitted, slurred, disarranged, or perhaps retarded or unduly emphasized. They are sharply distinguished from stuttering by the absence of sudden arrest of speech and by indications of paralysis rather than of spasm of the muscles of articulation. From aphasia they are distinguished by the fact that the arrangement of words is never changed, nor is the number deficient save in the terminal stage of extreme cases, and then owing solely to inability to produce the elementary sounds, not to defective word memory. The word anarthria may be used to designate any of these defects.

Stammering.

This word is here restricted to the sense that Kussmaul gave the German word *stammeln*, translated stammering in the English and

American editions of Ziemssen's Cyclopædia. It is unfortunate that the word is also used as a synonym of stuttering; we very much need an English equivalent of *anarthria literalis*, and it is simply a disadvantage to have two words for stuttering. In stammering, owing to difficulty in making the motions necessary for the production of some of the elementary sounds, these sounds are omitted or blurred or have others substituted for them. All children stammer more or less while learning to talk.

When stammering persists beyond the usual age for the acquisition of distinct speech it may be due to some purely mechanical defect, such as hare-lip, cleft palate, short frænum, or adenoid vegetations. In such cases the proper surgical treatment should be adopted without delay, for the longer stammering lasts the more likely is it to persist as a bad habit after the original cause has been removed. Many children stammer, however, without any organic cause. These patients have never learned the proper movements to make in order to produce the more difficult sounds and consequently substitute easier ones for them. In such cases intelligent and systematic teaching of the proper movements may do wonders in a short time. The child must be shown where to place the tip of the tongue in making the sound of th, t, d, l, and r, how to raise the back of the tongue in making k and g, how to place the lower lip against the upper incisors in making f and v, and so on.

As an acquired symptom stammering may be caused by any disease bilaterally affecting the fibres of the facial and hypoglossal nerves, the motor nuclei in the medulla, the tracts connecting these nuclei with the cortical centres for the vocal organs, or the cortical centres themselves. Unilateral disease may also cause dysarthria, but it is of brief duration, unless the motor-speech centre be involved. When the disease is in the cerebral hemispheres the utterance of some words may be altogether lost and then motor aphasia and anarthria may be combined in any proportion. When the left internal capsule is the seat of the lesion, as in ordinary right hemiplegia, there is first subcortical motor aphasia; then anarthria is apparent as the aphasia begins to disappear; as further progress is made towards recovery the anarthria alone remains and in a favorable case it also disappears. In left hemiplegia from lesion of the right internal capsule there is no aphasia, after the indirect effects of the lesion have passed away, but there is commonly some thickness of speech which disappears in a few days.

The typical stammering of organic disease is observed in bulbar paralysis, a disease in which, owing to a slow degeneration of the nuclei, the muscles of the lips, tongue, palate, and larynx are gradu-

ally paralyzed and wasted. In this disease, to use the vivid description of Kussmaul, "we perceive consonants and vowels successively crumbling away, as it were, from the patient's speech, while his intellectual powers may be perfectly retained. His words grow more and more indistinct and mutilated, his stammering passes into an unintelligible muttering, until at last he is only able to emit grunting noises, and perhaps not even these. So long as his hands and arms escape paralysis he continues to communicate his feelings and ideas by writing." The first difficulty is usually in articulating the lingual consonants, l, r, n, t, and d. As the lips become weaker whistling and the production of the sounds o and oo, in which the lip must be thrust forward, become difficult or impossible, and at the same time the labial explosives, p and b, and the nasal labial, m, are lost. When the palate is paralyzed, and so fails to shut off the nose from the mouth, all explosive sounds are impossible unless the nose be held, and the voice has a disagreeable nasal quality. As the muscles of the larynx become affected the voice grows hoarse and finally gives place to a mere whisper.

Syllable-Stumbling.

This is the characteristic speech defect of paretic dementia. It is particularly noticeable when the patient attempts to say words and phrases in which l and r are repeated; thus "truly rural" is apt to become "truly rulal" or "tuly lulal;" "the third artillery brigade" may be changed to "the third artrillerary bri-g-gade." When the patient fixes his attention upon what he wants to say and makes a special effort he is more likely to succeed, just the contrary to what is true of stuttering. Tremor of the lips or twitching of the face may accompany the effort to speak. As the disease advances other consonants are mixed or omitted so that speech may become quite unintelligible. Speech may also be disordered in paretic dementia by absence or perversion of ideas, true aphasia may occur, and stuttering may complicate the syllable-stumbling, but these are distinct symptoms. Syllable-stumbling is probably due to degeneration in Broca's convolution interfering with the production and coördination of the literal sounds into syllables but not changing the number or arrangement of words. It stands between aphasia and ordinary stammering.

Scanning.

In disseminated sclerosis articulation often becomes difficult and the first effect of this is to make the patient, by a special effort, pro-

duce the syllables slowly and with undue distinctness. becomes "ar-til-le-ry," instead of "artrillerary," as in pareti tia. At the same time the voice is monotonous and the ut described as scanning because it is like the mechanical sca Latin or Greek poetry by schoolboys. A certain amount of especially towards the end of a word, may be combined with ning, and in some patients the slurring occurs alone. As th advances scanning may give way to syllable-stumbling, lik paretic dementia, or to ordinary stammering, like that paralysis.

Aphasia.

Aphasia is an inability to properly receive or communic caused by disease within the cerebral hemispheres but n capacity of the ideation centres themselves. In all its fo distinguished from other disorders of speech by the fact th (considered as heard, spoken, read, or written) are deficient i or defective in arrangement.

Any organic or functional disease capable of interfering operation of the language centres or their connecting tracts r aphasia. The disease is always in the left hemisphere handed patients and in the right hemisphere in left-handed few cases apparently contradicting this statement have b lished, but their number is not sufficient to overcome the tion of error or incompleteness in the observation. As righ persons greatly preponderate the forms of aphasia will be as they occur in such persons, with the understanding that t right and left are to be interchanged to make the descripti to the left-handed.

As almost the whole of the cerebral apparatus for langu the cortex or immediately beneath it, so softening from th or embolism, which is the most frequent disease of the corte the most frequent cause of aphasia. Hence aphasia as a of vascular disease is presumptive evidence that the disease ing. Hemorrhage, however, may occur in the membranes part of the cortex or in the subjacent white matter, and t cause of aphasia; it is the most common cause of subcortic aphasia. Congestion without hemorrhage or partial occlusi artery not sufficient to occasion softening is the cause of s transient aphasias. Direct injury fracturing the skull, c the brain substance, or rupturing a vessel, is a very importa Inflammation of the membranes, by damaging the cortex or ing a reflex inhibitory effect, may cause aphasia just as i

paralysis. A cerebral abscess may lie beneath one of the language centres, and, by thus revealing its precise situation, urgently call for operation, as in a celebrated case of Macewen's. Tumors are not very likely to cause aphasia except as a part of general brain failure, probably because their slow growth allows time for the education of the language centres in the opposite hemisphere; nevertheless, any form of aphasia may occasionally be caused by a tumor.

Degenerative diseases, such as paretic dementia and disseminated sclerosis, affect many regions of the brain at once and seldom cause any well-defined form of aphasia. The defects in speech most characteristic of these diseases have already been described. In some cases, however, the damage is so concentrated on one or more of the language centres that true aphasia is the result. The apoplectiform seizures of these diseases abolish speech for a time, as they do all other cortical functions, and some form of aphasia may remain after other faculties have been regained. In rare cases of lead poisoning aphasia may be one of the indications that the brain is seriously damaged.

Many purely functional cerebral disorders and a variety of toxæmic conditions may be accompanied by aphasia. Probably every one has noticed that when he is fatigued there is some inability to recall words (verbal amnesia), and also a tendency to utter wrong words (paraphasia), both being evidence of a slight degree of auditory aphasia. Exhausting diseases, such as typhoid fever, may have a much greater effect upon speech. The complete loss of all forms of words in the celebrated case of Lordat was a functional loss remaining after convalescence from a fever. In "Romola," George Eliot has given a description of functional alexia, in the person of Baldasarre, that is of especial interest considering that the book was published in 1863, when aphasia was just beginning to be thoroughly studied. As some of the functional forms of aphasia are of great practical importance they will be discussed separately after the forms due to organic lesions have been considered.

The forms of aphasia are best defined and named according to the particular language centre that is at fault; thus we have four forms: motor, auditory, visual, and graphomotor aphasia. Theoretically each of these forms may be cortical, subcortical, or transcortical, according to the situation of the disease in the centre itself, in the tract connecting it with the periphery, or in that connecting it with other cortical centres. The old terms, ataxic and amnesic aphasia, have never been satisfactory and should be altogether discarded. All forms of aphasia are in a certain degree both ataxic and amnesic, as Kussmaul remarked twenty years ago. Even the term sensory

aphasia, so useful in contrast with motor aphasia when V made his great discovery of the auditory centre and the effe destruction, should now be used only when it is intended to d both auditory and visual aphasia or either indifferently.

Symptoms.

The principal symptoms of aphasia require definitio proceeding to describe its special forms.

Paraphasia is a perversion of utterance in which wrong w substituted for the right ones in a sentence and wrong litera for the right ones in words. Paralexia is a perversion of aloud in which wrong words or elementary sounds are substi the right ones as in paraphasia. Paragraphia is a precisely perversion of writing.

Word-deafness is the inability to understand words althou are heard. The idea is not aroused because the centres for do not receive the proper association impulses from the centre. Verbal amnesia is the inability to recall the sound o although the idea is in the mind. It is the converse deafness; the auditory centre is disabled or the association i it should receive from the ideation centres are interrupted.

Word-blindness is an inability, caused by disease in or left angular gyrus, to understand printed or written words a they are seen. Alexia is inability to read. When due t blindness it is visual alexia; when due to disease of the centre it is auditory alexia; when it is a symptom of destru the motor-speech centre, the most common cause of alexia, it called motor-speech alexia.

Agraphia is inability to write. When due to lack of p coördinate the movements of the right hand necessary for with ability to execute ordinary movements, it is called agraphia or graphomotor aphasia. When due to loss of me the appearance of the words to be written it is called agraphia, but visual agraphia is a better term because more In auditory aphasia paragraphia may be so extreme that the will make no attempt at writing, and we then have auditory a The most common form of agraphia, as already explained, is speech agraphia.

Apraxia is the inability to recognize the nature or use of through any one of the senses. It is a greater defect than which it always includes; it probably never occurs except fro eral cerebral lesions. Apraxia has a number of varieties, according to the sense through which recognition fails. Min

ness, or visual apraxia, is the condition in which the sight of a familiar object does not cause it to be recognized. It includes word-blindness because words are more difficult to recognize than ordinary objects. In mind-deafness, or auditory apraxia, the sound made by a familiar object does not suffice for its recognition; thus the tick of a watch does not recall the idea of a watch, nor the tone of a violin the idea of the instrument. Mind-deafness includes word-deafness. Psychic anæsthesia, psychic anosmia, and psychic ageusia are the analogous inabilities to recognize objects through the senses of touch, smell, and taste.

Asymbolia, or asemia, is inability to receive or communicate ideas by means of any signs whatever. It always implies a grave intellectual defect in addition to total aphasia and loss of gesture language.

Motor Aphasia.

Motor aphasia is an inability to utter words, due to disease of the motor-speech centre (cortical motor aphasia) or of the nerve fibres connecting it with the motor nuclei in the medulla (subcortical motor aphasia), but not to simple paralysis of the muscles of articulation. Its typical form is that due to destruction of the centre itself. In such a case the inability to pronounce any words or the very limited number at command distinguishes it from any other form of aphasia. The loss is usually sudden and at first complete, all articulate utterance and even gestures being abolished. But as soon as the indirect effects of the lesion have passed away gestures, and, as a rule, a very limited power of articulation return. A single syllable or a word or two is often the only thing the patient can say, and it is used on all occasions, no matter how inappropriate. These recurring utterances are sometimes utterly devoid of meaning; at other times they are intelligible words repeated automatically. One of Broca's patients always uttered the syllable "tan," on attempting to speak, except when excited, and he then brought out the oath "Sacré nom de Dieu." This oath could never be repeated voluntarily nor could any of the individual words be uttered at will. Another patient always replied to a question with the senseless sound "cousisi," but when annoyed exclaimed "sacon," probably an abbreviation of the oath "sacré nom." These oaths are merely automatic expressions of emotional overflow, the words being quite devoid of any intentional meaning. Some patients use "yes" or "no" on all occasions, often in direct opposition to what is meant.

In some cases the recurring utterance seems to be the last thing the patient said, or was going to say, before the onset of aphasia.

Thus a woman, who told a cabman to take her to "Mrs. Waters" and was immediately afterwards stricken, had the recurring utterance "Missis;" that of a librarian was "List complete;" that of a girl taken while riding a donkey was "Gee, gee;" that of a signalman stricken at his post was "Come on to me." One of my patients at first used the word "water" for everything she wished to say, but a priest undertook to teach her to speak and began by counting. As a result she learned the word "one," which, displacing the word "water," remained her sole utterance for many weeks.

Sometimes the ability to sing the words of a familiar song along with the tune is retained. This power, like that of uttering oaths, is always automatic, the patient having no voluntary command over the individual words of the song. The power of automatic utterance is generally explained as due to the action of the rudimentary motor-speech centre on the right side.

In partial destruction of Broca's centre the loss is less complete, but there is still a very marked restriction of the number of the words that can be uttered, and these are brought out with an obvious effort. Stammering is then combined with aphasia.

In striking contrast to the loss of utterance is the patient's complete comprehension of what is said to him and the immediate recognition of his own errors. If inquiry is made it is found that he recalls the sound of many of the words he cannot say. It seems to him as though he were going to speak correctly, but the disordered motor apparatus brings out the wrong sounds. Some words, on the other hand, are not recalled at all, doubtless because the auditory centre cannot remember them distinctly without being reinforced by association impulses from the motor-speech centre.

Writing is even more affected than speech. Even the few words that can be pronounced cannot, as a rule, be written, although the patient can generally sign his own name. The frequent retention of the signature in this and in other forms of aphasia is, no doubt, because of the automatic nature of the process of writing it. Other words must be mentally pronounced when written; not so with the signature.

Imitative speech and reading aloud are, of course, lost in motor aphasia, but it is a remarkable fact that the understanding of printed and written language is usually lost also. This, as already explained, is due to the fact that in reading visible words serve merely as signals for the corresponding utterances. Although the utterances are not audible the motor-speech centre remembers them and helps to recall the proper sound memories in the auditory centre, which then sends out the necessary association impulses to arouse the ideas. Alexia is

present in the great majority of cases of cortical motor aphasia, but in some cases the ability to read with understanding seems to be retained. Many of these cases I believe to be like those of Trousseau, in which the patient seemed to understand what was given him to read but really did not. The tests employed in some of the cases were certainly not very searching. We must believe nevertheless that sometimes the ability to read actually is retained, and this is to be explained by supposing that in exceptional individuals the auditory memory is sufficient without reinforcement by the utterance memory. Kahler has reported one case in which reading was retained after destruction of both the auditory and the motor-speech centre. In this case, which I believe stands alone, visual words must have aroused ideas directly, as in deaf-mutes.

Recovery from cortical motor aphasia may be very rapid if the centre is only indirectly affected or slightly damaged. During the progress of recovery the stock of words that can be pronounced gradually increases and the stammering that mars them at first decreases. For a time there is often a tendency to repeat the last word said instead of a difficult one following it, and a similar tendency is seen in writing. Should the centre be completely destroyed recovery often does not occur at all, and if it does it is necessarily very slow, the time being measured in years rather than in months, for adult patients. It must, of course, be by the education of the rudimentary motor-speech centre of the right hemisphere. This takes place much more readily in children than in adults; other things being equal, the younger the patient the better the prospect of a return of speech.

Subcortical motor aphasia may be caused by a lesion damaging the conducting fibres anywhere between the centre and the bulbar nuclei. The most common seat of the lesion is the internal capsule, and the aphasia due to it is usually accompanied by right hemiplegia and always by some degree of paralysis of the face or tongue. When the indirect effects of such a lesion have passed away the patient, although unable to talk, is able to recall words perfectly, to give the number of syllables in the words he cannot utter, and to read and write, as we would expect from the fact that the utterance centre is in communication with the other cortical centres and hence free to do its part in all these processes. A more remarkable fact is that the ability to talk usually returns in a few weeks, although the accompanying paralysis may last much longer or even be permanent. As in most cases the conducting power of the damaged fibres cannot possibly have been restored, and as no such rapid recovery occurs when the utterance centre itself has been destroyed, it must be that this centre is still able to influence the vocal organs, but through

another channel. Accordingly we have from Gowers the exp that the speech processes are arranged as usual in the le speech centre, and that this centre, through the fibres of th callosum, influences the corresponding centre on the right properly innervate the vocal organs. Under the constant inf the well-trained left utterance centre the rudimentary right o to do this in a small fraction of the time that would be requ were left to itself as in cortical motor aphasia. This expla confirmed by cases in which, after recovery from aphasia du ease of the left hemisphere, a second lesion of the right inte sule has caused a second, and this time permanent aphasi also confirmed by the effect of lesions in the white matte diately beneath the utterance centre.

Such a lesion, although sparing the centre itself, is like off communication with the other hemisphere as well as bulbar nuclei, and if this be the case recovery is as slow as in motor aphasia. Such a lesion will also, as a rule, interf writing by cutting off the necessary association impulses f centre, but it may be so placed as to spare this set of fib permitting writing, while interrupting those to the centre other side, and so preventing or greatly delaying the return o

Transcortical motor aphasia is of doubtful existence. L reports a case in which the words used in spontaneous spe mutilated, the ability to recall the sound of names of familia was greatly impaired, and there was paragraphia, the diff writing being for the same words as in speaking. These sy he regards as due to interruption of the association paths the centres for ideas and the motor-speech centre, distingui from cortical motor aphasia by the presence of imitative sp the ability to read aloud. It is difficult to accept Lichthe planation of this case because the visual and auditory centre very important part of the group of centres for ideas, and th have been in communication with the motor-speech centre a by the retention of imitative speech and of the power of readir Moreover, there are other possible explanations. Cortica aphasia was not entirely excluded, indeed it very probably e first, and if it persisted in slight degree the vigorous associa pulses of imitative speech and reading aloud may have been s to arouse the motor-speech centre when those of spontaneous and writing were not altogether efficient. A still more plau planation is the assumption of slight damage to both the and motor-speech centres. This is supported by the patie bility to recall the sound of the names of familiar objects.

Auditory Aphasia.

This form comprises the disorders in the use of language caused by disease of the auditory centre or of the fibres connecting it with other parts of the brain. It is said to be cortical, subcortical, or transcortical according as the seat of disease is in the centre itself, in the incoming fibres from the ears, or in the association fibres to other cortical centres. The great majority of cases are cortical. In a typical case, one in which the whole of the centre is presumably destroyed and its functions not yet carried on by the centre on the right side, the symptoms are very extensive, including word-deafness, verbal amnesia, loss of spontaneous speech or paraphasia, paralexia, alexia, and agraphia or paragraphia. As not all of these symptoms are present in every case and as some of them are found in other forms of aphasia, the following account of them is based on thirteen cases in which a fairly satisfactory autopsy showed the auditory centre to be diseased while the visual and motor-speech centres were spared.

Word-deafness is the most constant and most characteristic symptom of auditory aphasia. The inability of the patient to understand what is said to him cannot fail to make a deep impression on the examiner. A request to put out the tongue, shut the eyes, or cross the knees elicits no response unless accompanied by an explanatory gesture. When an inquiry as to his age has elicited no intelligible reply, the suggestion of different ages may be equally ineffectual; the patient not only fails to select the right one but he shows no surprise when asked if he is one year or a hundred years old. Either a strained, over-attentive, and anxious expression or a look of complacent fatuity may be a further indication that the words have no meaning for him. If the damage to the auditory centre is but slight the degree of word-deafness may be correspondingly slight, but it is probable that a careful examination would reveal this symptom in every case of auditory aphasia. In the thirteen cases in which a satisfactory autopsy showed that the first temporal convolution of the left side was diseased without involvement of the visual or motor-speech centre, word-deafness was present in eleven; in the other two, strange to say, no mention is made of the understanding of speech.

Verbal amnesia, or the inability to recall the sounds of words, is the converse of word-deafness. In the former case the diseased auditory centre acted upon by the association impulses from the centres for ideas, is unable to revive the memory of the sounds, while in the latter the diseased centre acted on by the impulses from the

ears is unable to send proper association impulses to the ce ideas. The inability to recall words is in most cases onl; and then it is found that certain classes of words are more be forgotten than others. Proper names go first and af common nouns. Verbs remain much longer, while pronc prepositions are generally used to the last. There can be lit that this difference depends on the difference in the freque which individual words have been used. With a few ex proper nouns are infrequently used in comparison with othe hence they are the ones most easily forgotten. The whole of common nouns in use by an individual is much greater whole number of verbs, hence one noun on an average is no often as one verb and is more easily forgotten. On the otl a very few pronouns and prepositions being used over and correspondingly fixed in memory. Verbal amnesia like word probably exists in all cases of auditory aphasia, but it has noted in so large a proportion of the cases. In the thirt already mentioned this symptom was noted in seven; in t six there is no definite account of the power to recall words. the inability is but slight, careful tests are necessary to ma parent. It must be remembered that this symptom also visual aphasia, and that motor aphasia is marked by loss of t memories of words which loss involves some impairment of t memories.

Some writers think that complete destruction of the centre would involve complete loss of spontaneous speech. loss, however, is very rare in auditory aphasia, while parap perversion of speech, is very common. In most cases the p of a considerable store of words is in strong contrast to meagre store or the recurring utterances of the motor aphasic is peculiar about the utterances of an auditory aphasic is tl many words are used and often pronounced correctly, some lated, and all are so mixed up together that in a typical patient's speech is an unintelligible jargon. In other ca idea of the meaning can be obtained from the patient's utte in the case of Starr's patient who said "five of telephone," "four of spades," or in the case of one of my patients who wife to "put some ice-water on the fire." Such patients are unaware of any absurdity in what they say; they are word-dea own speech as well as to that of others. Paraphasia is not a symptom of lesion of the auditory centre. Of the thirte already mentioned, it was present in six; in another it wa at first but afterwards disappeared; in another the patie

very little but was not paraphasic; in one case the patient did not speak at all; in the remaining four speech was good, although word-deafness was present in all of these. The absence of paraphasia in so many cases probably depends upon the incompleteness of the damage to the centre and upon the education of the right auditory centre.

Reading aloud in auditory aphasia is usually affected in the same way as speech; it is paralexic. Of eight cases out of the thirteen already mentioned in which reading aloud was tested, it was lost in one, although speech was good in that case; there was paralexia in four cases, and in all of these except one (who had recovered from it) there was paraphasia; in one the patient read only a very little, but that little was not paralexic; in two the ability to read aloud correctly was retained although neither patient understood what he read, and one had lost speech while the other was paraphasic. The ability to read aloud correctly in a patient who is both word-deaf and paraphasic and does not understand what he himself reads, is a very remarkable phenomenon which can be explained only on the supposition that in the reading of such patients the association impulses from the visual centre which pass directly to the motor-speech centre are sufficient to insure correct utterance without the aid of impulses from the auditory centre. The frequent occurrence in auditory aphasia either of paralexia or of total inability to read aloud shows, on the other hand, that when most persons read aloud the auditory centre does an essential part of the work, the memory of the sounds suggesting, as it were, the correct utterance, without which suggestion the motor memory would go astray.

The ability to understand written or printed language is commonly lost in auditory aphasia. In the six cases, out of the thirteen mentioned, in which this ability was tested, it was found to be lost in three, bad in one, slightly impaired in one, and good in one. These differences seem to depend mainly on the extent of the lesion.

Finally, the ability to write spontaneously is apt to be impaired in auditory aphasia. In the six cases (out of the thirteen) in which writing was tested, and there was no question of motor agraphia, it was lost in two, there was paragraphia in three and writing was good in two, the same two in which the understanding of written speech was retained and which were also free from paraphasia. Here, again, the differences seem to depend on differences in the extent of the lesion.

Of pure subcortical auditory aphasia we have very little precise knowledge, and I know of no case in which a satisfactory autopsy confirmed the theoretical interpretation of the symptoms. The following case from Lichtheim will serve as an illustration of this form:

A man, aged 55, had an apoplectic stroke causing paraphasia and paragraphia from which he almost completely recovered, a slight slowness of speech and an occasional false word being the only symptoms left. Five years later a second very slight stroke occurred after which he gradually became absolutely word-deaf, hearing remaining good when attention was directed to it. Spontaneous speech was fluent and correct, and there was no paragraphia, although writing from dictation was of course lost.

This and similar cases of word-deafness, without verbal amnesia, paraphasia, paralexia, or paragraphia, are readily explained on the supposition of a lesion interrupting the auditory fibres before they reach the left auditory centre, the integrity of the centre itself accounting for the absence of other symptoms than word-deafness. Freund has shown that bilateral disease of the internal ear may cause a certain degree of word-deafness which he calls "speech-deafness." Strictly speaking, this interesting condition is not a form of aphasia and may be distinguished from subcortical auditory aphasia by the presence of signs of disease of the labyrinth, such as dizziness, tinnitus, limitation of the auditory field, and marked loss of bone conduction.

The existence of pure transcortical auditory aphasia has not been confirmed by autopsy. A good clinical example of this form is given by Lichtheim (Case III.). A patient after a vascular lesion showed word-deafness but repeated the questions he did not understand. Spontaneous speech was fairly good, but there was distinct paraphasia and the corresponding paragraphia was still worse. Writing from dictation was good for single words but bad for sentences. Reading aloud was accomplished with difficulty but not understood.

The distinction between this case and one of cortical auditory aphasia consists only in the retention of imitative speech, and in view of the many possible combinations of symptoms due to partial lesion of the centre itself it is hardly safe, without the confirmation of an autopsy, to accept Lichtheim's explanation of an interruption of the paths IA.

Cases of combined subcortical and transcortical auditory aphasia are more likely to occur than pure cases of either form, but I know of no such case in which the centre was not also damaged or subjected to pressure.

The "conduction aphasia" (*Leitungsaphasie*) of Wernicke and Lichtheim, if it really exists, must be considered a partial transcortical auditory aphasia. The supposed lesion is an interruption of the path AM, which, theoretically, would cause paraphasia, paralexia, and paragraphia without word-deafness. Both of Wernicke's supposed examples of this form seem to have really been cases of visual aphasia; for in one hemianopsia was certain and in the other

highly probable; in one at least spontaneous speech was marked by amnesia of nouns rather than by paraphasia, while in both alexia and agraphia replaced the paralexia and paragraphia of the theoretical form. Lichtheim's example shows the symptoms required by theory, except that there seems to have been some defect in articulation not altogether paraphasic, but the autopsy showed both the auditory and motor-speech centres to be somewhat damaged, and it is quite possible that the symptoms might be caused by this damage alone.

Recovery from cortical auditory aphasia is usually much more rapid than from cortical motor aphasia. It is probable that, while this may in part be due to the greater extent of the auditory, centre it is mainly owing to the right auditory centre having a greater capacity for education than the right motor-speech centre.

Visual Aphasia.

Visual aphasia comprises the disorders in the use of language caused by disease of the visual centre for form (left angular gyrus) or of the fibres connecting it with other parts of the brain. As now known it is either cortical or subcortical according to the location of the disease in the centre itself or in the white matter of the occipital lobe. Transcortical visual aphasia is theoretically possible but has not yet been demonstrated.

The lesion of cortical visual aphasia must, of course, damage the cortex of the left angular gyrus and its immediate neighborhood. The disease often extends forwards into the supramarginal convolution. The subjacent white matter seems always to be damaged to some extent, thus accounting for the hemianopsia or the contraction of the right halves of the visual fields, which is always present. The lesion of subcortical visual aphasia is so situated as to leave the cortex of the angular gyrus free, but to interrupt the left optic radiation or destroy the left half-vision centre, thus causing hemianopsia, and at the same time, by involvement of the posterior fibres of the corpus callosum, to prevent the left angular gyrus from being reached by impulses from the right cerebral hemisphere. In this way the disease prevents the left angular gyrus from receiving new impressions of form, but leaves it free to remember impressions made in the past and to act on other centres in the left cortex.

In both the known forms of visual aphasia the ready utterance of many words and the full understanding of speech show that the auditory and motor centres with their connecting fibres are not at fault, while, on the other hand, right hemianopsia, or contraction of the right halves of the visual fields, makes a visual defect certain.

As word-deafness is the characteristic symptom of auditory apha-

sia so is word-blindness or visual alexia, the characteristic sign of visual aphasia; but as word-deafness is rarely pure, so word-blindness is usually accompanied by other symptoms due to the same cause. To a patient who is word-blind the written or printed words of his own language are, like those of an unknown foreign tongue, devoid of meaning, not because they are not seen well enough but because the necessary association processes from the visual to the motor and auditory centres do not take place. This defect may be absolute, in which case reading aloud is impossible, and all written or printed characters are devoid of meaning; but, as a rule, some words or characters are better comprehended than others. Thus the patient may name individual letters and not the word which they compose, or he may name certain familiar words and not their component letters. Figures are generally better comprehended than either letters or words. If the right hemisphere is intact, objects may be perfectly well recognized although words are not, for objects arouse the memory of their associated qualities through either visual centre, while associations with words have been established only in the left hemisphere. But mind-blindness sometimes accompanies word-blindness, and then, if an autopsy is obtained, a lesion is found in both hemispheres.

Although the auditory and motor-speech centres are intact in pure visual aphasia spontaneous speech is generally impaired. The principal defect is an inability to name objects seen. Thus when the patient is shown a knife he may be quite unable to name it, although his gesture shows that he fully understands its use. This inability is readily explained when we remember that the sight of an object arouses the memory of its name only through association impulses from the visual centre to the auditory and motor-speech centres. In an uncomplicated case objects not named at sight may be promptly named when they impress some other sense; thus a knife may be named when it is taken into the hand, or an apple or orange when it is tasted or smelt. Here, of course, another association path than that from the visual centre is brought into action, and, as a lesion limited to the visual centre or its neighborhood does not affect the other paths to the auditory centre, there is no difficulty. Were the auditory centre diseased, naming objects would be equally difficult no matter by what sense the object be perceived. In some cases an object is not named as soon as seen, but after looking at it a little while the patient names it without the aid of any other sense. In these cases it is an associated sense memory instead of an actual sensation that revives the memory of the missing name. The patient, for example, looks at an orange and fails to name it, but presently the

memory of its taste and odor is revived by association impulses from the right visual centre to the olfactory and gustatory centres, and these send the necessary impulses to the left auditory centre. The more the visual component of a word's meaning predominates the less likely is it to be recalled in this indirect way.

Closely allied to the difficulty in naming objects is the absence of nouns in spontaneous speech. As far as nouns are needed to name objects within the speaker's field of vision, this is only one phase of the difficulty already considered, but objects not seen but merely thought of are also not named. This is mainly because the visual component is the most prominent in the idea of most objects, so that the association impulses necessary to recall the name habitually start from the visual centre; but the less frequent use of nouns than of other parts of speech, already alluded to in explanation of the absence of nouns in auditory aphasia, must also be a factor here. The loss of association impulses from the visual centre is rarely absolute. Some objects are usually named at sight, and some nouns are recalled by the memory of the corresponding objects. Often the association impulses recall a word similar in sound or related in idea to the right one and then speech becomes paraphasic. The inability to recall names causes the patient to try to express his idea in roundabout phrases, as when a knife is called "something to cut with," and then speech is apt to become very much involved, and the paraphasia to be very marked. In some cases of visual aphasia, however, spontaneous speech appears to be wholly unaffected, without the lesion of the visual centre or of its connecting fibres being less than in other cases. I know of no satisfactory explanation of such cases, unless we assume that the right visual centre excited by the sight or memory of an object sends impulses first to the right auditory centre and thence by the corpus callosum to the left auditory centre, thus recalling the name.

In cortical visual aphasia, writing, whether spontaneous or from dictation, is often lost or impaired, and this is no doubt due to the fact that as one writes all but the most familiar words (such as one's own name) are visually represented, so that the coördinating centre for the hand is under the guidance of association impulses from the visual centre. If the visual memory of the word to be written is lost the proper motions cannot be made. This inability to write is known as sensory agraphia in distinction from motor agraphia. In the interest of precision it should rather be called visual agraphia, since auditory aphasia may be accompanied by agraphia which is also sensory. The ability to copy writing is also impaired for the same reason, but often to a less marked degree, because the actual sight of

the copy brings a stronger stimulus to the diseased centre than the mere memory of it. Unless the right visual centre is also diseased, so as to cause mind-blindness, the appearance of objects and pictures can be much better remembered than that of words. In subcortical visual aphasia, on the other hand, copying is impaired while writing from dictation is not necessarily affected at all, and spontaneous writing may be as good as spontaneous speech. Copying is lost because the lesion interrupts the impulses from the copy before they reach the left half-vision centre, and on their way from the right visual centre to the left visual centre (angular gyrus), consequently the left visual centre is not influenced by the copy. Spontaneous writing is retained because the left visual centre, being unaffected, can remember the appearance of words and properly influence the graphomotor centre.

The retention or loss of spontaneous writing is the principal means of distinction between subcortical and cortical visual aphasia and is of the greatest practical importance, because if an operation is to be considered it is necessary to distinguish sharply between the two forms. There can be no doubt that the ability to write while copying is lost is a very strong indication that the lesion has spared the angular gyrus, and must be sought deeper in the occipital lobe, while the presence of agraphia indicates that the lesion is in or near the angular gyrus. Nevertheless too much stress may be placed on these indications; a lesion in the angular gyrus might cause alexia and not agraphia, owing either to its small size or to an individual peculiarity of the patient, making his writing relatively independent of his visual memory, and a lesion deep in the occipital lobe may cause agraphia by its indirect effect upon the angular gyrus. In many cases of subcortical visual aphasia the retention of writing and the inability to read are brought into the most glaring contrast by the inability of the patient to read what he himself has just written; such a patient may say "I know what it is, but I can't read it."

In these cases another remarkable phenomenon may occur; the patient, by retracing what he has written, may be able to read it from the motions of the hand, and if his hand be moved by the examiner so as to trace words he reads them. In short, he reads only by writing. Such patients read writing more easily than print because it is easier to trace it, but one of Charcot's patients was aided in reading print by making the motions of writing each word. The explanation of these cases is not difficult on the assumption that the centre for visual memories is intact and in communication with the other speech centres, while more or less cut off from the half-vision centres. The motions of the hand vividly recall the appearance of the words written, and this suffices to recall the sound, utterance, and meaning.

Combined Forms of Aphasia.

Combined forms of aphasia are of frequent occurrence, because all the centres for speech are supplied by branches of the same artery, the middle cerebal. The combination of motor and auditory aphasia, due to lesion of both the third frontal and first temporal convolutions, is easily recognized clinically by the presence of word-deafness together with absolute speechlessness, or the very restricted utterances of motor aphasia, instead of the paraphasia which is characteristic of a lesion of the auditory centre alone. Reading and writing are affected even more than in auditory or motor aphasia alone. As a rule they are entirely lost, but if the damage to the motor centre is relatively slight paralexia and paragraphia may be present.

The combination of auditory and visual aphasia is recognized clinically by the presence of word-deafness and paraphasia together with defect in the right half of each visual field, alexia and either agraphia or paragraphia according to the situation of the visual lesion. When motor, auditory, and visual aphasia are all combined there is absolute loss of spontaneous speech or paraphasia (according to the degree of damage to the motor centre), word-deafness, alexia, agraphia or paragraphia (according to the situation of the visual lesion and the degree of damage to the motor centre) and defect in the right half of each visual field.

Dyslexia.

Dyslexia is a difficulty in reading characterized by the ability to read only a few words at one time on account of the rapid onset of painful fatigue. After a rest the patient can go on for a few words from where he left off, but must then stop. The seat of the disease is still a matter of pure speculation, but it is not due to any recognizable visual defect, nor accompanied by any definite form of aphasia. It is considered here because it seems to be allied to visual aphasia. It is generally agreed that this symptom occurs in patients whose arteries are degenerated, and that it is ominous of softening or of degeneration of the brain due to insufficient blood supply. Pick thinks that it is analogous to the intermittent limping described by the veterinarians as occurring in horses and by Charcot as occurring in men. A horse affected by this disease after trotting a few minutes begins to limp; if urged further it falls with signs of pain and the affected limb is found to be pulseless, cold, and anæsthetic. A rest restores the limb to an apparently normal condition, but the symptoms, which are evidently due to spasm of the main artery of the limb, return at the next attempt to trot. Precisely similar symptoms

occur in men, and in both horses and men these symptoms are usually forerunners of complete obstruction of the main artery of the limb. If we suppose the left middle cerebral artery to be diseased and to be thrown into a spasmodic condition by the fatigue of reading, the curious phenomena of dyslexia would be accounted for.

Graphomotor Aphasia.

Graphomotor aphasia, or motor agraphia, is the inability to write with the right hand, depending on loss of the power to execute the proper movements, without paralysis of the hand or arm. This definition applies to only a very small proportion of the cases of agraphia. In the majority of cases agraphia is a symptom of motor aphasia, and in almost all of the remaining ones it is a symptom either of auditory aphasia or of cortical visual aphasia. In 1881 Exner, on the strength of four cases, advanced the theory that the foot of the left second frontal convolution has the same relation to the centres for the simpler motions of the hand and arm, located in the Rolandic convolutions, as Broca's convolution has to the corresponding centres for the simpler motions of the vocal organs, and that, as destruction of the left third frontal causes loss of the complicated movements of speech without paralysis, so destruction of the left second frontal convolution causes a loss of the complicated movements of writing without paralysis. Since that time evidence has accumulated tending to prove the correctness of this plausible view, but as yet it is insufficient. In the first place there are no cases of complete motor agraphia. A patient entirely free from motor, auditory, and visual aphasia, who has learned to write, can always make the motions of writing, if not with the right hand then with the left, or with some other movable part of the body. Therefore the second frontal convolution can be considered a writing centre only in so far as it may be related to the special skill and ease with which the right hand executes the motions for writing. That it has such a relation seems probable, but I know of no case of pure agraphia in which the autopsy showed a small lesion of the left second frontal convolution and no damage to any other part of the brain. Any case in which the lesion affects the motor-speech centre is not conclusive, even though utterance be retained, because it takes a slighter degree of motor aphasia to interfere with writing than is required to prevent utterance. This is shown by the fact that in recovery from cortical motor aphasia speech may be recovered long before writing is, and, but for the history, such a case might easily be regarded as a case of motor agraphia. On the other hand, if the arm centre in the Rolandic region be affected the case cannot be used in proof of Exner's theory, for then a degree of paralysis sufficient to prevent the delicate

movements of writing cannot be excluded. Pitres' case, in which a man wrote well with the left hand, but with the right could only roughly copy, is not conclusive because there had been right hemiplegia and aphasia, and, although the patient is reported to have recovered from all symptoms except the agraphia, it is quite possible that the right hand and arm were still so impaired, as a result of the hemiplegia, that the remembered form of the letters was not sufficient for their production, while the stronger stimulus from an actual copy was. The case of the younger Charcot, while giving support to the theory, is also not conclusive. A woman after her first apoplectic attack had no symptom except motor agraphia. Years later a second attack left motor aphasia, and after repeated new attacks, there was pseudobulbar paralysis. At the autopsy a focus of disease was found in the foot of the left second frontal convolution, another in the foot of the left third frontal, and three in the middle of the right hemisphere. It seems very probable that the foci, occurring in the order named, caused the symptoms, but it does not appear from the autopsy record that the one in the second frontal convolution was older than the others and this leaves the matter in some doubt.

The practical question is: How far can agraphia alone be used as a localizing symptom in a case in which operation is to be considered? It seems from all the evidence now accessible that, given a case of inability to write with the right hand while writing with the left is retained and no paralysis or other form of aphasia can be detected, if the lesion is of such a nature that an operation may be expected to give relief, it is proper to trephine over the foot of the left second frontal convolution and explore. If after all it turns out that the lesion is in the motor-speech centre or in the motor centre for the arm, these centres are both immediately adjacent to the one exposed so that either may be reached without any very great enlargement of the original opening.

Functional Disorders of Speech.

Hysterical aphonia is the most common hysterical affection of speech. It consists in the inability to speak in a loud voice on account of the vocal cords not being approximated in the effort to produce sounds. As a consequence the patient speaks only in a whisper, but articulation is generally perfect. The loss of voice usually comes on suddenly during an hysterical attack or immediately after a violent emotion. In most cases there is no disease localized in the larynx, but sometimes a laryngitis or other localized disease may precede and be the suggesting cause of the aphonia, in which

case the onset is more apt to be gradual than sudden. The functional character of the loss is clearly shown by the perfect approximation of the cords in coughing and sneezing. A careful examination always reveals the presence of the physical or mental stigmata of hysteria. Gilles de la Tourette calls especial attention to the frequent existence of an anæsthetic area in the skin over the larynx, in accordance with the general law, which he has established, of the superposition of sensory loss upon hysterical paralysis or spasm. The interior of the larynx is often anæsthetic.

The diagnosis, which is generally easy, is based on the absence of a sufficient local cause, the ability to cough and sneeze, the circumstances attending the onset, the presence of other signs of hysteria and (in a few cases) talking aloud in sleep or when under the influence of an anæsthetic.

The prognosis is favorable. The voice may return at any time, especially after an attack or during excitement, and if it does not the patient gets along fairly well. The return may be gradual, but is usually sudden or very rapid. The time aphonia may persist, however, even under intelligent treatment, varies from minutes to years.

The great aim of treatment, as in all cases of hysteria, should be to reëstablish self-control. A favorable prognosis and confident demeanor combined with any harmless local treatment, such as mild electrical applications to the larynx, will generally restore the voice in a short time. Sometimes etherization, with or without a sham operation, succeeds brilliantly, but it is not without risk of making the patient worse. In one of my hysterically mute patients, who remained aphonic after careful teaching had restored articulation, etherization accompanied by predictions of cure caused a major hysterical attack and a relapse into absolute mutism. A return to pedagogic methods again restored whispering speech in about a week.

Hysterical mutism is far less common than aphonia, but has an intimate relation to it, so that one often shades into the other. In this condition even whispering is absent and the patient is unable to utter a single word. As the disease is undoubtedly in the cerebral hemispheres this mutism is a true motor aphasia, although purely functional. It differs from organic motor aphasia in two important respects. In the first place the loss is more complete than in organic motor aphasia. Not even the few words, such as yes or no, of the motor aphasic are used by the hysterical mute; with rare exceptions the loss of words is absolute, and so is the loss of voice. In the second place the intelligence of the hysterical mute is generally lively; he is anxious to communicate his ideas, his gestures are animated, and, most important

of all, he grasps paper and pencil and writes fluently about his symptoms. This is the general rule, but it must be noted that there are exceptions. In a few cases writing is lost just as in organic motor aphasia, and in one the utterance "ta-ta-ta" was retained; in another "how, how, how," in a third "yes" and "no." Hysterical deafness may be added to the aphasia, and then we have a spurious deaf-mutism. In one case, the exquisitely hysterical character of the affection was shown by the patient's ability to talk and hear from six to nine each day. Promptly at nine o'clock the larynx seemed to close with a peculiar noise, and both speech and hearing were gone for the day. According to Löwenfeld, a certain degree of word-blindness may also accompany the motor aphasia.

In a typical case of hysterical mutism the diagnosis is quite easy; the only speech defect that it could be mistaken for is subcortical motor aphasia in which writing is also retained. The complete loss of articulation and voice together with the absence of unequivocal signs of organic disease and the presence of some of the stigmata of hysteria are abundantly sufficient to make the difference apparent. Should writing be lost and some power of articulation retained, the diagnosis would be far more difficult. But even in such a case the difference between the accompanying symptoms and similar ones due to organic disease with the presence of hysterical stigmata would be a sufficient guide to an attentive observer.

The prognosis is both good and bad. Speech will return and may return at any time; but when, is uncertain, and relapses are very common. Aphonia usually remains as a transition stage between the aphasia and recovery, and stammering and stuttering in various combinations may also either precede or follow mutism.

The treatment must be that appropriate to any case of severe hysteria with the addition of such local treatment as will tend to restore the patient's confidence in his recovery. A great deal can be done by showing him how to place the lips and tongue and how to manage the breath so as to produce the consonants mechanically. When once a start is made in this way the progress is often very rapid, and I cannot help believing that in any case of hysteria educational methods which appeal rationally to the patient's own powers are far less likely to be succeeded by a relapse, or by other hysterical phenomena, than methods which, by appealing to his sense of the mysterious powers controlling him from without, may succeed very brilliantly for a time, but do nothing towards establishing a habit of self-control.

Stuttering, *stammering*, and various combinations of the two may exist in hysteria independently of mutism or aphonia. Spasm or

paralysis of the tongue or lips can often be detected, and case inspiratory spasm was the cause of the difficulty in It is impossible to give a precise idea of the disturbance of tion on account of its variable form in different cases or eve same case. The literal sounds may be omitted or slurred or sp cally emphasized or prolonged or variously disarranged, anc adventitious sounds may be interjected. The treatment h sufficiently indicated.

Epilepsy.—Epileptic attacks may be preceded, as an a motor aphasia, and probably by the other forms also. St common is it for aphasia to remain as a transient after-effec attack, in the form of motor, auditory, or visual aphasia, o bination of all. Pick has carefully studied the re-evolution o in such a case. In the epilepsy symptomatic of any organic which is susceptible of relief by operation a well-defined aphasia is a valuable localizing symptom. A purely hysteric aphasia may succeed a genuine epileptic attack just as an h seizure may.

Migraine accompained by ophthalmic disturbances may al companied by aphasia which is always transient and portends than that the migraine will be especially refractory.

Chorea.—Chorea when severe commonly interferes with which is at first made indistinct by the irregular movement tongue and lips. This causes the patient to hurry and short ance as much as possible, and then, perhaps, to become al mute. As there can be no doubt that the cortex is the seat of in chorea it seems safe to conclude that the difficulty is cause ritation of Broca's convolution, and hence is a true aphasia. my choreic patients who seemed keenly observant of all t going on about her, and certainly understood all that was said presence, would not even move the head to indicate yes or no.

Fevers.—Typhoid fever, especially in children, is not very cause of motor aphasia, which is mostly purely functional and without signs of organic disease, towards the end of a seve often when the other symptoms are abating. It is then no ominous symptom and always disappears with returning s Far more serious are the rarer cases, occurring in adults a often as in children, in which hemiplegia accompanies the and which are due to embolism and consequent softening quoted by Wylie). Such a complication adds greatly to the of death, and if the patient recovers both the hemiplegia and are likely to persist, except in young children, in whom t hemisphere is more easily educated. Dysarthric disturbanc

also occur in typhoid fever and are of more unfavorable significance than functional aphasia. Pneumonia and other fevers may occasionally cause similar affections.

Toxic States.—States of autointoxication, particularly uræmia, diabetes, and gout, and poisoning by various narcotics or by snake bite occasionally cause aphasia or dysarthria.

Deaf-Mutism.

The scope of this work does not call for a full discussion of deaf-mutism, but its relations to other forms of aphasia are too close to allow the subject to be entirely omitted or only casually mentioned. A deaf-mute is a person who, owing to congenital or early acquired deafness, does not talk. The earlier statistics indicated that a very large proportion of the patients were congenitally deaf. This was no doubt an error, as many causes of deafness in early life are often entirely overlooked by parents, and a child is supposed by them to have been born deaf unless it has given unmistakable evidence of hearing. Any bilateral disease of the ears or of the auditory centres or connecting tracts may be a cause of the deafness. Heredity, consanguinity of parents, and residence in certain mountain regions are the principal predisposing causes. Epidemic meninigitis, scarlatina, typhoid fever, measles, and other fevers are the principal exciting causes. The mutism depends entirely upon words not being heard and so not imitated. When deafness is congenital or occurs during the first year of life, speech is not acquired at all; when it occurs in the second, third, or fourth years what speech has been acquired, although it may have been very considerable, is in almost all cases forgotten, unless unusual methods of education are employed. Deafness occurring after the fourth year causes mutism in a certain proportion of cases, which rapidly diminishes as the age increases, becoming very small after the tenth year, and vanishing altogether at puberty. Those deaf-mutes who have ever acquired speech, even if it be totally forgotten, have a great intellectual advantage over those who have never acquired it.

Intelligent deaf-mutes invent for themselves a gesture language which serves for ordinary communication with those nearest to them. By association with others this language is very much extended and elaborated, and as it contains signs for abstract as well as for concrete ideas, it serves very well as an instrument of thought instead of the words which ordinary people employ. In addition to the sign language it is not very difficult to teach them reading and writing. The process of reading differs from that of normal indi-

viduals, because the written word cannot br
sponding word as heard and uttered, and so
representing it, must be associated directly
the word. Writing is also different, becaus
first calling forth the sound and utterance of
their visual appearance, which suggests the
writing them. When reading and writing l
manual alphabets (of which a number have be
telegraphic code, and various systems of shor

Of late years it has been common to teac
ing the pupil carefully observe the motions of
and chest which are necessary for the produ
then try to imitate these motions. After so
this mechanical process has been obtained, th
the lips of another and uttered by the pupil
with the ideas they represent either directly
words. Those who have once had hearin
succeed far better than others in learning
even in the totally and congenitally deaf the r
education are very surprising. Nevertheless
mains, if one may say so, the patient's mot
vehicle not only for communication with his
thinking. The words of normal individuals,
language, are like a foreign one, in which
write, and even think, but from which he cons
into the more primitive and to him more nat
It is mainly on this account that even the b
show a strong tendency to avoid the society of
seek that of their fellows.

The Diagnosis of Disorders

This is not the place to consider the diag
which cause the disorders of speech, since
elsewhere in this work. These disorders a
point of view, only a part of the symptoms
pathological diagnosis is to be inferred. But
peculiar and very complicated groups of sym
ways easily distinguished from one another,
to append a table giving in outline their m
clinically.

A. Spoken words are badly formed but (co
of the patient) not deficient in number or arr

last stages of bulbar paralysis, when the utterance of words may be altogether abolished. Reading and writing are not affected.

I. Speech is arrested by a spasmodic retention of some part of the vocal apparatus in one position, especially when the patient feels himself to be under observation—*stuttering*.

II. Certain literal sounds are formed only by an excessive effort, or not at all, or sounds are misplaced so that words are slurred, distorted or mutilated—*anarthria*.

B. Words (as spoken, understood, read, or written) are deficient in number and those spoken and written may be improperly arranged. Reading or writing or both are affected, except in subcortical motor aphasia, subcortical auditory aphasia, and hysterical conditions.

I. Speech heard and understood.

1. Articulation and phonation completely abolished. Reading and writing retained except in the rarest cases. No signs of organic disease. Stigmata of hysteria present—*hysterical mutism*.

2. The words spoken are very few or perhaps none at all, but the patient is not aphonic. The few words retained may be used on all occasions without regard to their meaning.

(*a*) Writing and quiet reading retained—*subcortical motor aphasia*.

(*b*) Writing abolished. Comprehension of written or printed language impaired—*cortical motor aphasia*.

3. The stock of words used is large, but proper and common nouns may be lacking. Inability to read aloud or to understand written or printed words. Acuity of vision not diminished enough to account for the alexia. Some defect in the right half of each visual field, usually hemianopsia.

(*a*) Spontaneous writing retained. Copying impaired—*subcortical visual aphasia*.

(*b*) Writing abolished, owing to inability to recall the appearance of words as written—*cortical visual aphasia*.

4. Utterance and reading normal. Inability to write with right hand, owing to incoördination without paralysis. Writing with left hand retained—*graphomotor aphasia*.

II. Speech heard but not understood. Paraphasia, paragraphia, and alexia (paralexia on reading aloud) usually but not always present—*auditory aphasia*.

III. Speech not heard. On account of deafness patient has either never learned to talk or has forgotten how—*deaf-mutism*.

Relation of Disorders of Speech to Insanity.

1. As insanity constitutes no protection from the various disturbances of speech it is obvious that they may occur in an insane person, just as in a sane one, without having any special relation to the mental condition. As far as speech is concerned such cases require no discussion, but from the alienist's point of view a considerable difficulty in diagnosis is introduced by the difficulty of communicating with the patient.

2. Aphasia occurring in a person previously sane has a distinct tendency to enfeeble the mind by interfering with internal language, and hence with thought upon abstract or complicated affairs. This enfeeblement is generally not sufficient to be called insanity, but it may be of great importance. It is greatest in auditory aphasia, in a typical case of which verbal amnesia and paraphasia must introduce great confusion of thought as well as of speech. In motor aphasia the mental loss is much less, but it is always apparent to an observer who has known the patient before the loss of speech. The French neurologists have emphasized the importance of individual peculiarities in determining the amount of mental impairment in such cases. Some people in silent thought depend much more than others upon a vivid idea of the utterance of the necessary words, and if such persons be attacked with motor aphasia their disturbance of thought will be correspondingly greater. Uncomplicated visual aphasia, since it deprives the patient only of names which can usually be replaced by circumlocutions, has but little effect upon thought.

3. An organic disease causing aphasia generally has some deteriorating effect upon the mind irrespective of the loss of internal speech, and the impairment may be so great as to constitute insanity. Thus a cerebral vascular lesion or a tumor which destroys one of the language centres very often interferes, directly or indirectly, with the function of other centres which are necessary for ideas, and dementia is the result. Even when other cortical centres and their association tracts escape direct injury, the indirect irritating or depressing effects of the cerebral lesion may cause dementia or a maniacal or melancholic condition. Anarthria is not so commonly associated with mental impairment, and a patient quite speechless from bulbar paralysis may be perfectly clear in his mind; still the same degeneration which causes anarthria in paretic dementia, by affecting the cortex also, causes the mental disease, and a similar relation may sometimes be observed in disseminated sclerosis.

4. In various forms of insanity in which there is no special dis-

turbance of the apparatus for speech, there are disorders of speech resulting simply from the disorder of ideation. Idiots are often speechless because they have no ideas to express; in melancholia speech is slow, perhaps absent, on account of the slowness of ideation, absence of attention, and general depression characteristic of the disease; the rapid, continuous, and often incoherent speech of maniacs corresponds to the rapid flow of ideas and the general motor excitement; a paranoiac may talk continually or not talk at all in obedience to a supernatural voice, and so on. Such symptoms of mental disease are of the utmost importance to the alienist, but they can only be mentioned here; for a full description of them, the reader is referred to special treatises on insanity and especially to the work of Séglas.

5. The hallucinations of insane patients are often due to a special excitation of the language centres. The most common of these is that of a voice outside the patient, threatening or mocking him or perhaps echoing his thoughts. All sorts of delusions are based on such hallucinations, which no doubt result from an overexcitability of the left auditory centre. A less common hallucination is that of an internal voice which is not heard like other voices but is felt internally. Such imaginary voices may be located in the mouth, throat, stomach, abdomen, or other part of the patient's body, and a number of them may be present at once. Séglas has shown quite clearly that this hallucination is one affecting the motor-speech centre. Just as some patients have the hallucination that a limb is moved when it is not, so these patients have the hallucination that the vocal organs are moved without recognizing the movement as their own, but attributing it to an internal voice separate from their own. In some of these cases the lips and tongue are actually seen to move, and speech may even be quite audible, but the patient asserts that he has said nothing.

Hallucinations of seeing written or printed language, or of having the hand compelled by some external power to write what the patient does not wish to write, occasionally occur, but they are rare.

Medico-Legal Relations of Aphasia.

The question of criminal responsibility very seldom arises in reference to patients with aphasia, but it occasionally happens that the ability of an aphasic to make his will or to manage his property must be decided upon by a court. In the consideration of such a case the first question to be asked is whether the patient is insane irrespective of the aphasia. This is to be answered by the presence or absence

of such symptoms as would justify a diagnosis of insanity in a case without disorder of speech; the only difference in the manner of investigation is that introduced by the difficulty of communication. If the patient is insane, the speech defect can only add to his incompetency, and a decision is easily reached unless the form of insanity is such as would not in itself seriously affect general mental capacity, such as a limited and harmless delusion. The form of insanity most likely to accompany aphasia is dementia.

Should the patient appear to be sane the next question is: How far does the disorder in the use of language in itself affect his capacity to make a will, to enter into contracts, or to give testimony? Any form of aphasia has a certain tendency to diminish intellectual power and control of the emotions, even when there is no indication of actual insanity, and this must be taken into account as a factor distinct from the patient's difficulty in understanding others or making himself understood. The different forms of aphasia, however, differ very greatly in their effect on the mind, and different patients with the same form or the same patient at different times may show very great variations in mental capacity; so every case must be studied separately and entirely on its own merits.

Subcortical motor aphasia, after the indirect effects of the lesion producing it have passed away, has very little necessary effect upon testamentary or business capacity. The cortex being uninjured the flow of ideas and their formulation in internal speech are unrestrained; it is only their oral expression that is prevented and this is usually compensated for by the ability to write. In cortical motor aphasia it is somewhat different. The patient can neither speak nor write and so can communicate with others only by gestures or by expressing assent or dissent when questioned. But notwithstanding these difficulties motor aphasics usually manage to make themselves well understood by those intimate with them and often show considerable ability in playing games and in the management of concrete affairs. As they can readily understand all that is said to them and show plainly that they do understand it, their wills and contracts have usually been held to be valid. It should be remembered, however, that motor aphasia, by causing some words to be lost from memory altogether and by causing those retained to be revived in the auditory centre alone, thus making them less effective, must seriously interfere with thought upon any complicated subject. In the cases which have established the precedent, the affairs dealt with have been very simple, so that they might readily be thought of in concrete images without the aid of words. It seems to me that if a man had a large amount of property of different kinds, some of it in the abstract form of stocks and

bonds, and if there were a number of persons having just claims upon him, some of whom he saw habitually and others not at all, and if this man should be stricken with motor aphasia, the impairment of the ability to think in words and to read the papers representing his property would very probably prevent his fully realizing what he had to bequeath and the relative strength of the claims upon him.

Visual aphasia, since it does not interfere with the understanding of oral speech nor with the oral communication of ideas, has very little effect upon testamentary capacity; although the patient cannot read he can perhaps write and at any rate can converse, and thus make his wishes perfectly clear to those about him and readily be reminded of considerations which if left to himself he might forget.

With auditory aphasia it is very different. In a typical case all forms of communication are interfered with. The patient cannot say what he wants to on account of paraphasia and paragraphia; he cannot understand what others wish to say to him on account of word-deafness and alexia. Moreover, the ability to think in words is interfered with far more than in motor aphasia, owing to the verbal amnesia and paraphasia of internal speech. Thus it is not only impossible, as Gowers says, to know what the patient really wants, but it is probably impossible for him to consider his own wants except those of the simplest character. Slighter degrees of auditory aphasia might not, of course, wholly exclude testamentary capacity, but any case in which there is word-deafness and enough paraphasia to make speech uncertain in its meaning should be regarded as one of incompetency, unless gesture language is very clear in its meaning and the matters to be considered are very simple in their nature and limited in extent.

Deaf-mutes were in ancient times regarded as irresponsible just as though they were insane, but the modern view is very different. Many decisions have established the doctrine that educated deaf-mutes may give testimony, make wills, enter into contracts and dispose of property just as freely as other persons. In theory, they have also been regarded as fully responsible for criminal acts. In practice, uneducated but intelligent deaf-mutes have been held to a full accountability as far as theft and other crimes against property are concerned, but as to crimes against the person, such as assault and homicide, when committed under provocation, courts and juries have made such a distinction that the offender has either been punished very lightly or not at all. Aside from the influence of ancient precedents upon the legal mind, the reasons for such leniency are apparent.

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I

THE DISORDERS OF SLEEP.

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THE DISORDERS OF SLEEP.

General Considerations.

SLEEP or its equivalent is a function common to a large part, if indeed not the whole, of animal life. Many ingenious and conflicting theories as to its actual causation have been elaborated, and many difficult and painstaking experiments have been carried out for the purpose of determining precisely the various phenomena which immediately precede, follow, and accompany this function. However imperfect, unsatisfactory, and even contradictory these speculations and experiments and the deductions from them have been, they comprise the existing physiology of sleep and must be discussed to some extent before the disorders of sleep can fairly be considered.

The ultimate causation of sleep is probably as unknowable as is the origin of force, and therefore need not be further considered in this place.

While there is a wide variation among healthy individuals in the normal exercise of this function, it is convenient to erect a somewhat arbitrary standard of healthy sleep, broad enough to embrace all these variations, and to study the phenomena therein contained, to the end that deviations from this standard, or disorders of sleep, may be understood.

The most common and essential characteristic of normal sleep in the adult is the abeyance of consciousness, which implies a suspension of the most important functions of the neurons of the cerebrum. During waking moments these are in a constant state of varying activity, modified by stimuli which are all the time conveyed to them from the environment by the afferent nerves connected with general and special sensibility. This functional activity gives rise to ideas which are often permanent and have a correcting influence on the formation of new ideas resulting from subsequent stimuli received from the environment; and finally these permanent ideas or memories may by an effort of the will be recalled and compared with each other, with the result of the formation of still other ideas. This last process, in which stimuli from the environment play little or no part, is generally designated as cogitation, and has to be reckoned with as one of the most troublesome features in many cases of insomnia.

In the act of falling asleep, either consciously or unconsciously, the will often plays an important part. The individual wishing to sleep seeks a place as free as possible from external stimuli and by an effort of the will endeavors to disregard any that may remain, and by a similar effort abandons or banishes cogitation and disposes himself so that there may be complete relaxation of the voluntary muscles, thus relieving the cerebrum of the stimulus known as the muscular sense.

Within a variable period, during which some slight change of position may be made until one habitual to him is assumed—probably most compatible with complete muscular relaxation and easy functionating of the various viscera—sensory stimuli are less and less attended to, ideas become more and more vague and confused, and in a delicious but fading sense of general well-being consciousness dissolves and sleep supervenes.

Without discussing their significance at length it is proper to state that pronounced changes in many of the bodily functions are regularly observed during natural sleep, differing from those which occur in the waking state; but at this point a warning ought to be given against confounding these changes with the causes of sleep. While it is a fact that all or nearly all of them result directly from alterations in the nervous system itself incident to sleep, in the present state of knowledge it would be difficult or impossible to establish such a relation, and therefore it is better in describing them to adhere to the divisions commonly adopted by physiologists.

It is easily demonstrated during sleep that the heart beats less strongly and the number of contractions decreases on an average from ten to twenty per minute while the general arterial tension is considerably lowered.

The changes which take place in the circulation of the brain during sleep have been studied in children by observing the fontanelles; in adults who have survived an accident or surgical operation, by which much of the skull has been removed; and in the lower animals by removing portions of the skull and dura mater and fixing glass in the openings. It has been quite conclusively demonstrated from the above methods of investigation that during sleep the brain shrinks very perceptibly, diminishes much in vascularity, and that these conditions are more pronounced in sound than in disturbed sleep. In fact, like other organs—the stomach for example—the vascularity of the brain varies directly with its functional activity. This contraction of the cerebral vessels is synchronous with a corresponding dilatation of the vessels of the arms and legs, and so sensitive is the mechanism controlling these variations that a ray of light falling on

the eye or a slight sound is sufficient to cause contraction of the vessels of the legs and arms with a correspondingly increased vascularity of the brain.

The theory that coma is analogous to natural sleep and that each condition was caused or accompanied by cerebral hyperæmia was at one time quite popular, but now I think has been pretty generally abandoned.

The respiration averages four less per minute during sleep than when the person is awake and partakes more of the thoracic type. This function is normally performed with considerable irregularity during both the waking and sleeping moments, and in either condition may normally present in some degree the so-called Cheyne-Stokes rhythm.

Whatever may be the significance of it, experimenters agree that during the twelve hours of the night considerably more oxygen is absorbed and considerably less carbonic acid eliminated than during the twelve hours of the day.

The fall in temperature is from 0.2° to 0.5° F., and is usually greatest shortly after midnight, while the amount of heat given off is less than half as much during the sleeping as the waking hours.

Secretion, excretion, digestion, assimilation, and intestinal peristalsis are all less active during sleep.

The foregoing phenomena appear the same in those who work by night and sleep by day, though for some reason not well understood few individuals who thus transpose their hours of repose and work sleep as well, even if the bedroom be ever so quiet and carefully darkened, as when the natural order is observed. Such persons, with rare exceptions, finally become nervous, present well-marked signs of disordered health, and have to return, for a time at least, to nocturnal sleep.

In sound natural sleep the pupils are contracted and the cremasteric, plantar, abdominal, and tendon reflexes are absent, which is a confirmation of the view that the peripheral (spinal) as well as the central (cerebral) neurons are functionless; for were the cerebral neurons alone functionless and the spinal neurons remained active, then an exaggeration of the reflexes might be expected similar to that which obtains in cases of hemiplegia resulting from cerebral hemorrhage.

Sleep probably reaches its maximum intensity at about the end of the first hour and continues for several hours when fluctuations begin to appear and increase until waking occurs.

Lepine and Duval have recently put forward a very plausible hypothesis regarding the condition of the neurons during sleep and the waking moments respectively. Each claims priority over the

other of a few weeks in the announcement of this theory, wh sentially that the protoplasmic processes of the neurons amœboid movements, contracting during sleep in such a mar produce a functional isolation of each of them and expandi on waking so that all previous relations between them are Ramon y Cajal has likewise put forward an hypothesis wl poses a similar alternating contact and separation of the but he assumes it to be due to a corresponding contractior pansion of the neuroglia cells. At any rate, to assume th ation and contact do occur as above indicated conduces g clearness of views in the contemplation of many of the fun nervous matter, and without arguing the point the theor adopted in this article simply as a working hypothesis.

In dreamless sleep it may be assumed that all the neurc in the waking state participate in processes of thought bec connected from one another. Dreams are accounted for posing that for some reason certain neurons or groups of do not fully contract, and thus incomplete cerebration conti sensory stimulus from the stomach, for instance, after a he per pretty generally causes an expansion of and contact bet neurons in which certain memories are stored, without wa sleeper, and he dreams accordingly. Exposure of the part may provoke dreams of wading in cold water, and similarl bling noise may be associated by the dreamer with a thund In these latter cases it is not difficult to understand how th mentioned might cause sufficient expansion of the appropriate to give character to the dreams. Nor according to this the difficult to understand why the neurons that are much exerci frequent and prolonged fixation of the mind on any giver should expand somewhat during sleep and so determine the dreams. Moreover, it is not unreasonable to suppose that ex should occur over a very irregular and limited area, giving ri most painful, varying, and chaotic dreams that can be con when no very apparent cause for such expansion presents it might be expected, too, that individuals would, within norma present marked differences in the character and extent of their

Finally, according to this hypothesis, if it happens tha neurons concerned in volitional movements and many of th narily concerned in intellection in the waking state expand, bu remain contracted and disconnected so that the individual is conscious or, more properly speaking, fails to recognize identity, then the condition of somnambulism is accounted f somnambulist is far more awake than asleep; he has failed

awake. Somnambulism is certainly not a phenomenon of natural sleep, and is alluded to in this place partly for the purpose of amplifying the hypothesis I have adopted. For the same reason hypnotism may be briefly mentioned in this place. In certain stages, at least, of this condition the reflexes are increased—that is, the peripheral neurons remain active—while the influence which the central (cerebral) neurons normally have over them is withdrawn—that is, some of the central neurons are contracted. The hypnotized subject is able voluntarily, upon the suggestion of the operator, to produce such contraction of the central neurons as to present many of the phenomena of sleep, while at the same time he may sustain certain of the neurons, as, for instance, those required for innervation of the muscles employed to hold the arm a long time in a constrained position, in a higher and more durable state of expansion than he would be able to do in his normal state. At the suggestion of the operator he so acts upon his neurons as either to fail to appreciate at all or to interpret falsely many kinds of strong sensory stimuli.

A healthy person on awaking from the number of hours of normal sleep which is natural to him feels fresh and buoyant both in body and in mind, and resumes the work which perhaps he had quitted the night before in weariness and discouragement with hopefulness and zest. The mathematician, the inventor, and the business man alike often achieve their most brilliant results rapidly or even suddenly on awaking in the morning, before having time to get out of bed. Problems over which they have previously spent many earnest and perplexing hours are solved in a moment without the least conscious effort; and not infrequently with the morning the fallacies of the conclusions reached and the plans laid before retiring, when mind and body were fatigued, are only too apparent.

Thus sleep is undoubtedly a recuperative process in which the whole system participates, though the effect is most manifest on the nervous system because this system exerts such a strong controlling influence over all the other bodily functions. Alternating periods of activity and repose, waste and repair are common properties of organic matter, and especially organic matter endowed with a nervous system. If this alteration does not take place in a fairly definite time the organism perishes; it must be regarded as inherent, and the presence of this or that substance in the tissues, or its absence from them, does not account for it. As already suggested, logical reasoning upon the question of the prime cause of sleep soon merges into the question as to the origin of life, matter, and force, which are beyond mortal ken.

It is well known that sleep normally varies considerably in

accordance with age, sex, temperament, occupation, season, and climate.

The healthy infant, if not disturbed, sleeps almost constantly during the first few weeks of life, and the hours spent in sleep gradually diminish till the age of twelve or fourteen, when nine or ten hours are sufficient, and this amount is generally needed till the period of adolescence is reached; from maturity till the time when the influence of advancing years becomes manifest, eight hours are sufficient; while old people often doze a great deal, they appear to require rather less sleep than is taken in middle life.

It is generally stated that women require more sleep than men, but that for a time at least they bear loss of sleep better; the latter part of this statement, however, lacks confirmation, as the evidence upon which it rests is by no means conclusive. Under existing conditions of civilization, the duties incident to maternity cause more or less disturbance of sleep, covering a period of many years of adult life; and then, too, the nursing of sick people, involving much loss of sleep, in a vast majority of cases is performed by women, and indeed it often does seem surprising that they bear loss of sleep so well; but a considerable experience with professional male nurses has tended to convince me that they under the same conditions bear deprivation of sleep as well as women do.

The best guide as to the time required by an individual is that he sleeps till he feels refreshed. It is certainly a mistake to curtail the morning sleep of a growing child in order to make him conform to certain methods of housekeeping. If discipline has to be applied in this direction, a retiring hour should be insisted upon which would insure enough sleep.

The quality as well as the quantity of sleep is of importance. People not infrequently complain of feeling dull, heavy, and unrefreshed after eight or nine hours of profound and dreamless sleep; they not inaptly term this unnatural sleep, judging it by the after-effect. It is a matter of common experience, too, though difficult of explanation, that one may awake in the early morning fully refreshed after five or six hours' sleep, but if he fall asleep again for an hour or two, he awakes feeling miserable, and often does not recover himself for several hours.

The modern tendency in medicine to reduce everything to an exact scientific basis has resulted in the neglect of many useful and practical doctrines. Thus the doctrine of temperament has been almost entirely neglected of late by authors and teachers, though that it has some practical utility cannot be denied. The subject of temperaments cannot be discussed here further than to note that persons of the neu-

rotic temperament commonly take comparatively few hours of sleep and are specially prone to suffer from insomnia, while those of sanguine temperament suffer much less, and those of the lymphatic temperaments least of all from this affection.

An outdoor life with plenty of bodily exercise, plenty of good food and good clothing is more conducive than any other to the most perfect performance of all the bodily functions, sleep included. From the very nature of their occupations brain-workers are frequently irregular in their habits regarding work, out-door exercise, and sleep. On the whole they sleep less and are much more liable to suffer from insomnia than body-workers.

It is a matter of common observation and experience that sleep is more prolonged during the winter than the summer months, and similarly a cold climate is more conducive to sleep than a warm one. The climate of the temperate zones is the most highly conducive to the effectual performance of both bodily and mental functions, and it is in that zone alone that practically all the great bodily and mental achievements of man have been attained.

Insomnia.

The term insomnia is a very comprehensive one. It is a symptom, often troublesome and important, of so many different diseases and conditions that it becomes necessary in order to discuss it profitably to make some more or less natural or arbitrary divisions and subdivisions of it.

Two broad classifications at once suggest themselves. The first would comprise all the cases not associated with any of the well-recognized somatic or mental diseases, and the second would embrace the cases which are so associated. The former may be designated functional and the latter symptomatic insomnia. Functional insomnia will be considered first.

By functional insomnia is meant in this article, in addition to the definition already given, a considerable abridgment of the sleep natural to the individual, extending regularly or irregularly over a period of some days at least.

Etiology.

Predisposing Causes.—This disorder is comparatively rare under the age of eighteen in females and twenty-five in males, probably because prior to that time forces of development are still to some extent in operation, and more especially because at about those ages the various responsibilities and relations of life have to be met and sup-

ported. If the cases incident to the climacteric period and to are not included, by far a majority will be found to occur betw ages of twenty-five and fifty-five. A much greater number o however, is met with subsequent to this period than prior to deed, some of the most typical and inveterate instances of the c that have come under my observation have become confirmed after fifty and lasted with some brief and partial remissions f to twenty years.

Sex does not appear to be of much etiological importance ing that the occupations of men are of a nature much more l produce insomnia than are those of women.

Temperament and heredity may conveniently and logically sidered together in this connection, as they are so intimately ated; their influence is often conspicuous. There is no better e to be found in support of the law of heredity than that the association of characteristics known as temperament is fre transmitted either directly or indirectly from progenitor to p and it may be confidently asserted that individuals in whom t rotic or neuropathic temperament predominates are especiall to suffer from insomnia, and indeed nearly all other functic orders of the nervous system. They undertake a great de quently work under high pressure far into the night, and hard of or feel the necessity for sleep till this function has beco ously impaired; whereas the lymphatic individual easily re his excellent sleep habit if force of circumstances compels hi time to abandon it.

Exciting Causes.—Certain occupations and the habits of th lowing them, frequently act as exciting causes of insomnia. eral terms it may be stated that mind-workers suffer much mo insomnia than body-workers; but while this is undoubtedly t reason for it lies deeper than at first sight is apparent. The sition needs explanation lest the influence of mere bodily exe overestimated. The principal exciting causes of functional i are anxiety and long-continued hours of close mental appl with insufficient time allowed for sleep. When these two ca continued, the result is often speedily disastrous. The farm spends his days in the open fields and whose hours are not long, nor whose labor is unduly hard, finds for some reason cannot meet his obligations and is likely to lose the home he ha years in making, constantly has before his mind ways and m salvation, with fears of failure and its consequences. He beco victim of insomnia, his regular out-door life and bodily exerc withstanding. The student, the literary, the professional, a

business man suffer from insomnia usually because mainly of prolonged and continuous mental effort, with or without anxiety, and not on account of a lack of bodily exercise. Indeed, they could not well add the bodily work of a day laborer to their own, and if they did so, their liability would be thereby increased rather than diminished. The assertion merely that a man is a body-worker to some extent warrants the inference that for some reason he is not a mind-worker and therefore not subject to insomnia; nevertheless, if for any reason not including grief or anxiety the laborer becomes idle and lives under the same conditions as to bodily exercise as the brain-worker, he sleeps as well as ever. The relation of bodily exercise to sleep will be referred to again when the treatment of insomnia is discussed.

Nursing a sick relative when, in addition to interference with regular hours of sleep, there is a more or less constant state of anxiety with perturbation of the feelings, is highly conducive to insomnia. This cause, of course, is much more frequently met with in women than men, because this work ordinarily falls to the lot of women. If, however, the elements of sympathy and anxiety are greatly reduced or eliminated, as in the case of professional nurses, the tendency to insomnia becomes correspondingly modified.

Enthusiastic brain-workers of good physique often discover that they can by habit reduce their hours of sleep so as to take only two or three hours in twenty-four without feeling any serious ill effect; this may go on for months—rarely for years—a person meantime accomplishing a prodigious amount of mental work, and congratulating himself upon the success of his method; indeed, he may sincerely conclude that his case is not exceptional and that the majority of mankind might, if they would, safely practise a similar habit. Sooner or later, however, even the little sleep that he has so grudgingly spared the time for is denied him and he falls a victim to the terrible tortures of one of the most unrelenting and intractable forms of insomnia.

Finally it not infrequently happens that individuals in all walks of life, in whom no predisposing or exciting cause to account for the trouble can be discovered, suffer from a pronounced attack of functional insomnia.

There is a considerable number of exciting causes, properly designated as insanitary, which tend to impair the general health without producing definite disease, such as badly ventilated rooms, insufficient food or clothing, and unwholesome occupations which lie near the border line separating essential from symptomatic insomnia.

Whatever may be the cause of insomnia, in most cases, sooner or later, symptoms of neurasthenia are often associated with it; but while these symptoms not infrequently become prominent before the insom-

nia appears, it is doubtful if they should be regarded as causes but rather as disturbances of function due to the same causes as the insomnia.

Symptoms.

While the symptoms of functional insomnia present wide variations, most cases naturally fall under one of two divisions: either the sufferer experiences great difficulty in falling asleep, or he falls asleep promptly on retiring and awakes in an hour or two to find that every vestige of somnolence has vanished. In the former instance the mechanism which normally brings about contraction of the neurons and diminishes the cerebral circulation does not act promptly and cerebration continues; moreover, in consequence of this, cerebral influence is not fully enough withdrawn from the voluntary muscles and these are not properly relaxed, comparatively slight sensory stimuli continue to be appreciated, and therefore the individual frequently changes his position in order more completely to relax now this and now that set of muscles in an effort to find complete and lasting ease. The subjects that have engrossed the mind by day continue to occupy it; the same neurons are mainly the seat of the disorder, but others may also continue to act though in a modified manner, hence the ideas formed in this state are generally notoriously lacking in clearness and reliability, a fact that the patient himself often appreciates. Finally, when sleep does supervene though consciousness vanish, many groups of neurons continue to display considerable activity, which is manifested by dreams and general restlessness. In the less severe cases, after a longer or shorter period, the neurons pretty generally contract and sleep becomes less disturbed, and may be even quite sound and refreshing for a few hours before rising time. In more severe cases it frequently happens, unless hypnotics are given, that the whole night passes without consciousness having been entirely lost. It is in this form of insomnia, however, that patients are apt to overestimate their disorder, for if a careful watch is kept over them it will be perceived that though they snore for a period of five to twenty minutes several times during the night and present other unmistakable signs of sleep, they feel positive in the morning they have not slept a wink.

The form of insomnia now under consideration almost always has an obvious exciting cause, and in a great majority of cases this is found to consist of mental overwork and worry. In severe forms, if relief is not found, an acute attack of insanity is to be apprehended, and I have seen not a few cases culminate in pneumonia (frequently fatal) or acute articular rheumatism. Sometimes almost from the first there is considerable digestive disturbance, constipation, rest-

lessness, and irritability of temper; but in all (even moderately severe) cases some of these symptoms usually make their appearance along with others. Patients generally become greatly discouraged and depressed and complain that both by night and by day their mental agony is beyond endurance, so that not infrequently relief is sought in suicide. So long as the patient is able to give some attention to his daily duties these distract him somewhat, and towards and during the evening his mind may be easier so that he himself is surprised at the contrast in his feelings. This marked improvement in the feelings as the day wears on—which may continue until the patient often feels almost as well as ever in the evening, having been exquisitely miserable in the morning, with perhaps vertigo and nausea—is one of the most common and conspicuous characteristics of neurasthenia; and, as already remarked, neurasthenia almost invariably accompanies insomnia, though the converse does not hold, for it often happens in neurasthenia that sleep is deep and sufficiently prolonged, though not refreshing.

The capacity for taking food is sometimes preserved especially for the midday and evening meals, though it is not eaten with natural relish, but in the majority of the cases now under consideration the appetite is greatly impaired and the body weight diminished. Febrile disturbance is absent or insignificant.

Though they have many characteristics in common, that form of insomnia in which the patient falls asleep promptly on retiring and then wakes up in from one to four or five hours in a state of complete and lasting vigilance, has many points of difference from that just described. In this latter form the sleep, while it lasts, is generally natural and refreshing, and doubtless accompanied by normal changes in the neurons; but for some reason not understood these changes are not permanent enough, and the system does not have time properly to recuperate. It quite frequently happens that no adequate exciting cause can be found to account for the existence of this form of disorder; it appears to depend mainly on some derangement of the vital properties inherent in all organic cells; that is to say, it is pre-eminently functional in its nature. It generally develops gradually, either with or without some of the exciting causes previously mentioned. A person whose sleep had previously been satisfactory observes that he wakes up an hour or two earlier than had been his custom, notwithstanding he retires at his usual time, goes promptly to sleep, and sleeps soundly. He feels refreshed and wide awake, but, reasoning that he has not slept enough, tries vainly to fall asleep again; he feels no bodily discomfort except possibly a sense of general restlessness which he has to make an effort to overcome, and

remain in bed, while his mind is most aggravatingly fresh ar notwithstanding his attempts to suppress his mental activi exercise of will power. This involuntary and unwelcome ce is generally very different in character from that already re which begins immediately after retiring and prevents the pati going to sleep. It is frequently of very desultory nature ar means confined to topics which engrossed the mind during Incidents of the past sometimes quite remote come before and imperatively demand attention; the contemplation of tl or may not of itself arouse distressing emotions. When th comes to realize, however, that he is powerless to banish t sleep, that he has lost the most precious part of his liberty, fi self in fact powerless in the grasp of a cruel monster—and themselves often use a simile of this kind—his misery bec tense. He usually worries a great deal and becomes d retires, fearing and expecting that he will repeat his experie night before, and this no doubt increases not a little the pr of his doing so. It is a somewhat curious fact, for which I h been able to find a satisfactory explanation, that if the patie these circumstances attempts to postpone the waking hour b later, he meets with disappointment, for his sleep terminates the same time as before.

In cases which pursue an unfavorable course, the waking approaches the retiring hour till they are not separated by n an hour or two, or perhaps meet. Every now and then afflicted with this form of insomnia is encountered who asse tively that he has not lost consciousness in sleep for sever A clergyman, of about forty, once consulted me, who stated that he had not slept for nine months. More commonly sleep from one to three hours five or six nights in the week main awake the other nights. I have known people in ar beyond middle life who claimed to have gone on this way and who at the same time regularly did a moderate amount and presented a fairly good bodily appearance. Though all quite conscious, such patients often maintain the same pos several consecutive hours without discomfort, the pulse and tion decline somewhat, and it is not improbable that the undergoes some degree of recuperation. There is almost i some depression and sometimes it is severe, amounting t melancholia; in which case initiative, power of application, affection, interest in the affairs of life, and capacity for enjoy all reduced; the conventionalities of life are performed mec if not automatically, even to eating and drinking, etc., for t

appetite, and sensibility are dulled so that the comfortable sense of repletion which in health follows the ingestion of a good meal, or the relief which follows a free movement of the bowels is no longer enjoyed.

There is a strong inherent tendency to improvement or recovery in this form of disorder, most marked up to the first half of middle life, and there is not nearly so strong a liability to acute insanity (possibly simple melancholia ought to be excepted) or other disease as in the previously described form of insomnia. All cases are subject to recurrences and after fifty the disorder may persist with partial remissions of variable duration for years. Mild cases are very common in which for several days or weeks without any appreciable exciting cause the amount of sleep habitual to the individual is abridged nightly by several hours; this much more frequently happens during the summer than the winter months, but it is by no means confined to them. The influence of the early morning light is worthy of consideration in this connection, but in most of the cases which have come under my observation careful darkening of the sleeping-room has not been crowned with so much success as it ought to have been if this cause were really of much moment. If the patient does not become alarmed over his condition, it is frequently very surprising how slight may be the bodily and mental distress or discomfort resulting from a considerable loss of sleep in these cases; indeed, on this account patients sometimes form erroneous conclusions regarding the importance of sleep, and neglect treatment accordingly. Many people have several more or less severe attacks of this kind in the course of a lifetime, but recover spontaneously without any very serious results.

Symptomatic Insomnia.—Broadly speaking, in symptomatic insomnia either sensory impressions arising from disease or injury in some part of the body continuously act upon the brain, or, on account of disease, toxic matters accumulating in the blood act upon it in such a manner as to prevent those changes already described, which always accompany normal sleep, from taking place. A discussion of all the diseases which act in this manner does not properly belong here, but a few types may be mentioned, by way of illustration.

Disease giving rise to sensory impressions of a character to produce insomnia may be either acute or chronic and may cause various degrees of pain or mere discomfort. Furuncle, neuralgia, and peritonitis may be taken as examples of the former; dyspepsia and different skin diseases as examples of the latter. In these cases much depends upon the predisposition of the individual, for while at first the disease in question may have acted as an exciting cause, the function of sleep may become so much disordered as to demand primary

consideration. In fact insomnia, even when merely a symptom, demands much attention, for it exerts a most unfavorable influence upon every disease in which it occurs, chronic skin diseases not excepted.

Many acute febrile conditions, gout, lithæmia, and Bright's disease, may be cited as instances in which toxic principles circulating in the blood probably give rise to insomnia.

Pronounced insomnia is, as has been stated, a frequent prodrome of acute insanity, and even more frequently is it a prominent symptom of that disease; indeed, in a large proportion of cases it is the most urgent symptom. Sometimes it appears to be a potent etiological factor, but generally it may more properly be considered as one of the many evidences of disordered brain function which together constitute insanity.

It does not seem quite clear nor is it a matter of great moment as to whether the insomnia of insanity belongs properly in the functional group or not, for, as already intimated, the boundary separating symptomatic from functional insomnia is a very broad one, and many cases lie fairly upon it, now shifting a little more to this side and now to that.

Diagnosis.

As to the mere existence of insomnia the statements of patients have to be relied on, but much allowance has to be made for unintentional exaggeration, for even though a patient is placed under the careful scrutiny of a night nurse, very frequently he cannot be convinced that he sleeps as much as the nurse's report would indicate. Doubtless it sometimes happens that both are somewhat in error, though the patient, however, is very liable to claim that he has been only partially asleep when he has presented unmistakable signs of sleep for an hour or more. This is probably because the quality of sleep is somewhat changed; the obliteration of consciousness is not quite complete, the general sense of repose is lacking, and the refreshment following normal sleep does not come.

The main diagnostic problem consists not in deciding whether insomnia exists or not, nor yet as to the exact amount of curtailment of natural sleep, but as to whether it is functional or symptomatic and if functional, under which of the divisions already made it falls; if symptomatic, of what disease or disorder is it a symptom. This is often difficult because functional insomnia may culminate in insanity, and become symptomatic, or it may arise in the course and as a consequence of such a disease as neuralgia and persist stubbornly after its primary exciting cause had disappeared, that is to say, it would first be symptomatic and finally functional. And again, in such

a disease as gout, in which toxic substances present in the blood act on the brain so as to prevent sleep, this function may become so much disturbed that a habit is formed which persists in the absence of its original cause.

In order to reach a correct diagnosis in many of these mixed cases numerous obscure points in the etiology and symptomatology of a wide range of diseases have to be canvassed.

Treatment.

In the treatment of symptomatic insomnia the matter of foremost importance is the treatment of the disease of which the insomnia is a symptom. To discuss the subject from this point of view will not be attempted in this article. When, however, as often happens, insomnia becomes, as a result or accompaniment of other diseases or conditions, a symptom of such prominence as to demand special therapeutic attention, it has to be treated as a functional insomnia, and such modifications and compromises have to be made as the requirements of the fundamental disease may impose.

Functional insomnia resists treatment most obstinately. It has already been shown that natural sleep is something more than mere loss of consciousness, hence it follows that drugs which abolish consciousness are not necessarily hypnotics.

Returning to the hypothesis that during natural sleep all the neurons concerned in voluntary movement, general and special sensation, and ideation are contracted, and thereby in a measure separated from each other; that the vessels of the brain and probably of the spinal cord are contracted and the parts comparatively bloodless, and that in the rest of the body the vessels are dilated and contain more blood than during the waking state, it follows that the rational treatment of functional insomnia would consist in resorting to measures which most effectively produce these conditions with the least disturbance of the other bodily functions.

The advantage of adhering, in discussing the treatment of functional insomnia, to the division made at the outset is obvious when the etiology of the two conditions is remembered.

In the first form of functional insomnia the most obvious indication is to remove the exciting cause, for in nearly every case, as has been stated, one exists in the form of mental worry or overwork.

Where it is practical the mental diversion and relaxation incident to travel for a few weeks or months often effect a speedy and lasting cure without medication. After such an outing has been planned, the first night of the journey, spent perhaps in a train, frequently puts a

permanent period to the insomnia. A few days even of hu fishing may do much good towards giving the patient a start road to recovery, though in this connection the tendency to ought not to be forgotten, for the victim of this disorder is liab suddenly seized with a powerful suicidal impulse, and in s event it is very undesirable that he should find himself alone forest with a loaded gun in his hands.

Unfortunately, however, it is often far easier to convince tient of the benefits that would accrue to him by dismissing and taking his ease than to suggest a practical way for him t On this account, therefore, some sort of a compromise has to b If a period of absolute rest and diversion is not obtainable, tl worked business or professional man may be induced to inte quite frequent and definite periods of pleasant diversion w working hours, for it is the long continuous stretch of wo wears. An hour or more at the noonday meal with congeni pany is very helpful and the actual sacrifice often trivial. (exercise is of great value, especially if while taking it the pat joys full mental relaxation, otherwise its influence is much les

After everything practical has been done to establish goo of rest, recreation, outdoor exercise and work, and after suitabl ures directed to the general health have been instituted whe are indicated, if insomnia still continue, means must be res which are calculated directly to induce sleep. The sleepin should be well ventilated and the temperature, if artificial used, should be not over 60° F. So far as possible agreeable diversion and relaxation should be sought several hours be tiring. The effect of various devices to divert the mind from subjects and thus invite sleep is well understood by the laity; ample, certain kinds of reading best known to the patient, n nous, distant and soft musical sounds, counting, etc. These measures are sometimes useful in overcoming a tendency to in or as adjuncts to more positive treatment in developed cas their influence is not great.

In a few cases electricity appears to be useful, but to what suggestion plays a part is not easily determined; at any rate, it is advisable in employing electricity to associate as much sug with it as possible. When the patient is ready for bed, a galva rent of from two to five milliamperes may be passed through th for from five to ten minutes by placing an electrode three in diameter above and somewhat back of each ear; the polarity changed once during the *séance*, the current being turned of this is done. The current must be turned on and off very gra

otherwise vertigo and nausea may be produced. In any case this treatment has to be undertaken with great care, many patients being so sensitive to it that it has to be abandoned. It is best, however, if it is decided to try galvanism, not to warn the patient of the possible unpleasant results.

I have had no experience with hypnotic suggestion in the treatment of insomnia nor do I know of any reports based upon careful and continuous observation. That suggestion is a potent factor, however, in the successful treatment of many cases of disease is plainly recognized by every intelligent physician, hence it would seem rational to assert that this subject should be scientifically taught in medical colleges, but for some not very well understood reason both the public and profession regard with some degree of suspicion any practitioner who openly avows the confidence in it which experience and observation appear to warrant.

The influence of bodily fatigue is favorable to the induction of sleep, and a patient who has not already had too much exercise may sometimes with advantage take a brisk walk before retiring.

Non-medicinal measures, which are assumed to act by producing a determination of blood to various parts of the body and thus lessen the quantity in the brain and cord, may now be considered.

Massage, given in a way most agreeable to the patient, affords the advantages of exercise without an effort of the will; but more than this, it draws the blood to the surface of the body, thus tending to render the brain and cord to some extent anæmic as they are in natural sleep. When not obnoxious to the patient, it is often of great value.

Heat to the feet and abdomen is useful and well adapted to mild cases. Where available, the electric mat is the best means of applying artificial heat locally, for it is light and the proper degree of temperature can be maintained for any length of time without disturbing the patient. Next to this comes the Japanese stove, which consists of a metal box of suitable shape and size, covered with flannel or velvet, and heated by a fuel cartridge which burns for about an hour without odor. In the absence of these, hot-water bags, bottles, compresses, or even mustard plasters or poultices may be used. If agreeable a cold cloth applied to the forehead may heighten the effect of the other measures. Though less powerful than the warm bath or wet pack these means are often preferable to those because they involve less disturbance to the patient and may be steadily employed without producing prostration.

The warm bath should be at the temperature of from 90° to 100° F., and the patient should remain in it for from five to fifteen minutes, be speedily rubbed dry in the manner most agreeable to him and im-

mediately put to bed. When it seems desirable, the effect of the bath may be maintained by heat to the feet and abdomen.

The wet pack is given by placing a pillow on a mattress in a position to support the head, and spreading one or more large blankets over this, and then over these a sheet wrung as dry as possible out of cold water. Upon this the nude patient lies down on his back and an attendant folds one side at a time, first the sheet and then the blankets over him, puckering them properly about the neck and tucking them well in so that the sheet lies in close contact to the whole surface of the body, including the legs. The effect may be heightened by the application of heat to the feet and a cold compress to the forehead. As it is not the purpose to promote profound diaphoresis, comfort should determine the amount of covering used, and if the patient does not fall asleep in the mean time, after from thirty minutes to an hour, he should be rubbed down and put regularly to bed. If he fall asleep, the process of rubbing may be postponed till he awakes, the attendant seeing to it in the mean time that the covering is adjusted in conformity with any changes of position or temperature of the room.

Both the wet pack and the warm bath, if employed nightly, are decidedly "weakening," and therefore should rarely be resorted to more than three or four times a week. They often exert a powerful and salutary hypnotic influence.

The utility of food in the treatment of insomnia is often very decided. If stomach digestion is good, a glass of warm milk, or iced if greatly preferred, with or without soda biscuit, or in fact any other light meal of easily digested food will sometimes speedily be followed by sleep. This doubtless acts mainly by attracting blood to the stomach, thus relieving the brain and cord like the hot applications, the warm bath and the wet pack, but it should never be combined with these, for they attract the blood from the stomach to the surface of the body and thus indigestion results.

Alcohol in the form of beer, milk punch, or hot toddy taken at bedtime sometimes acts well. I can recall several cases, however, in which temperate business men who, under severe and somewhat prolonged anxiety, became insomnic and found that a "stiff" drink of whiskey on retiring would put them promptly to sleep; the anxiety continuing, the dose of whiskey was increased till nearly a pint was taken without the desired effect, and a few days later acute insanity developed.

In severe cases in addition to the foregoing measures medicinal hypnotics have to be employed.

In full medicinal doses the bromides diminish the reflexes and allay excitability and are thus conducive to sleep; the potassium salt will

be found the most generally satisfactory, but either of the others may suit particular cases better. Much mental depression or simple melancholia contraindicates their use. Given in conjunction with the measures already suggested, it quite often happens that no other medication will be needed; they may, however, in most instances be combined with advantage with the more powerful hypnotics.

Of the stronger medicinal hypnotics, chloral, somnal, sulphonal, trional, and paraldehyde are the best known and most reliable, and of these chloral stands first, though some of the others are formidable competitors. Hypothetically in addition to their other effects they may be assumed to produce contraction of the neurons. Opium demands separate consideration.

Chloral weakens the heart's action and respiration, lowers arterial tension, dilates the superficial blood-vessels, reduces the temperature, and produces anæmia of the brain; in fact it produces all the known phenomena of natural sleep. Sleep commences in from ten to thirty minutes after the administration of the drug and lasts from four to seven hours. Usually the patient awakes refreshed and rarely suffers any unpleasant effects, such as headache, nausea, or vertigo. It is no doubt true that death has occasionally resulted from its action on a diseased or weakened heart, but the danger from this cause has been overestimated and on this account chloral has been withheld and an inferior remedy employed, sometimes, no doubt, to the detriment of the patient. I am led to this conclusion from my asylum experience, because I have many times felt compelled to give large (thirty to forty grains) doses of chloral to insane patients with various forms of organic and functional heart disease and so far without an accident, and I am sure many other physicians in asylum practice could corroborate the opinion here expressed.

Chloral or any other medicinal hypnotic should rarely be used oftener than every alternate night, for it may happen that the effect is more marked on the night following than on the night of its administration. It not infrequently happens, too, that the effect of chloral is so happy that the patient insists on a nightly dose and discards all other measures of treatment, thus falling into imminent danger of forming the chloral habit. The proper use of these hypnotics is to assist in effecting a cure of insomnia and not as a makeshift, which may cause the patient to place a false estimate on the gravity of his malady. The taste of chloral may be disguised by syrup of orange peel or lager beer.

In general the sleep produced by none of the remaining drugs is so natural, so lasting, or so refreshing as that produced by chloral, while, as already stated, they act less promptly and certainly, though

they are all regarded as safer in cardiac weakness as they do not appreciably affect the circulation. Any one of them is fully as likely to be followed by unpleasant after-effects as chloral, but for various reasons, often not well understood, may be better suited to individual cases. Somnal in my hands has proved the most prompt and certain, while sulphonal and trional have seemed the best suited for protracted use as in cases of simple melancholia. Hypnol, a crystalline combination between chloral and antipyrin, has hypnotic properties, but has not been long enough in use to be assigned a definite place in therapy, and the same may be said of hypnone.

Apart from its anodyne properties the happy hypnotic effect which opium has in some cases of insomnia is not very obviously suggested by what is known of its physiological effects. Bearing in mind the special danger of formation of the opium habit and the contraindications afforded by badly diseased kidneys, it ought always to be tried in protracted cases in which medicinal hypnotics have to be employed, because its effect cannot otherwise be positively foretold. The insomnia and depression of simple melancholia sometimes vanish as if by magic before opium, and the same is true of the restlessness and insomnia of functional and organic heart disease and of senility. Sometimes one preparation of opium will succeed when another had utterly failed.

In the second form of insomnia the results of treatment are not so positive as in the first.

In the absence of an appreciable exciting cause the therapeutical indications are simply to maintain the general health in the best manner and to direct remedies to the re-establishment of the disordered function.

When sleep intervenes promptly upon retiring and lasts for three or four hours, the administration of hypnotics is rarely advisable, because very large doses are then required to produce the desired effect, and when rising time comes, the condition of the patient is manifestly worse than it would otherwise have been. In such cases it is better for the patient to make himself as comfortable as he can under the circumstances. If he is not easy as he lies in bed, he may divert himself by light reading and moving about his room, in the mean time taking a light meal, as a glass of milk or beer with soda biscuit; thus it will often happen after an hour or two he can rest comfortably in bed again, though he may not fall asleep. Indeed on retiring a second time, all the non-medicinal measures previously mentioned may be resorted to.

If the sleep is manifestly too brief, then a maximum dose of some hypnotic should be given on retiring, when waking will be postponed

frequently several hours. If this is done several times a week, needed rest will be afforded while the bad sleep habit will be broken in upon. For this purpose sulphonal has the advantage of other hypnotics in the fact that its influence is delayed for an hour or more after its administration. In pronounced functional insomnia the physician will have an opportunity of testing several different hypnotics, and even though he should find each of them equally well suited to the case, more or less frequent changes will be found to enhance the effect of each by preventing the establishment of a tolerance.

Insomnia of Insanity.

Insomnia is one of the most conspicuous of the complexus of symptoms which together constitute insanity, especially acute insanity. Though commonly spoken of as a symptom of insanity it is not strictly a result of this disease, and hence, according to the definition previously given in this article, it is not symptomatic as it might be, for instance, when resulting from neuralgia, but mainly functional. It may not be of particular advantage, however, in this place to insist too strongly upon the classification, for, doubtless, in many cases after acute insanity has developed it tends directly to interfere with sleep.

Though, as has been stated, either the first or the second form of functional insomnia may culminate in insanity, in the developed disease in nearly all cases the first form only is met with, the disturbed motility and mental derangement standing somewhat in the relation of an exciting cause and preventing the patient from going to sleep.

There is a considerable degree of insomnia in nearly all cases of acute insanity, but I have now and then seen a case in which, notwithstanding considerable mental and bodily activity, insomnia never became marked. In paranoia developing subacutely it may be entirely wanting, whereas in chronic melancholia patients often suffer from it for many long years. So long as the appetite is good, the vegetative functions are well performed, and the pulse is in good condition, there need be no great cause for alarm in a case of acute insanity, though there may have been no sleep for several days; indeed, in such cases natural sleep from exhaustion sometimes asserts itself and is accompanied by a rapid amelioration of all the symptoms, which may then go on to recovery or may be interrupted by other periods of insomnia and excitement.

Acute insanity, in the majority of instances, runs a fairly definite course of from two to six months, and as insomnia is very generally a prominent symptom throughout, it is highly important to avoid as far as possible anything like the steady use of medicinal hypnotics; because thus given, in order to produce the desired effect, the dose

has to be steadily increased till the injury done the nervous and digestive systems far outweighs the benefit.

After what has just been said it may be stated that the general principles already laid down for the treatment of the first form of functional insomnia are the ones to be depended on here, though in acute insanity there are a few conditions requiring special consideration. The coöperation of the patient cannot be reckoned on, hence more positive and potent measures have to be employed, either non-medicinal or medicinal.

When there are no signs of weakness to contraindicate it, an excited patient may generally be taught in a few days to walk with one or two attendants, and thus the influence of outdoor exercise may be had whenever and in such degree as desired.

The wet pack, too, is especially adapted for these cases, for, by the employment of strong safety pins, it may be made to act as a light form of mechanical restraint, which is, however, often well received, as its effect disposes strongly to tranquillity. With fatigue and the wet pack may be conjoined the medicinal hypnotics in severe and obstinate cases, but when thus used as an adjunct a much smaller dose will be found efficacious. Indeed, I have seen many patients pass safely through a very sharp attack of acute insanity from start to finish without a single dose of a medicinal hypnotic of any character.

During the first two or three weeks of an attack of acute insanity careful study with well-trained and conscientious attendants will often demonstrate that the case can be safely managed without the use of medicinal hypnotics; that after several consecutive days and nights of complete insomnia, natural sleep occurs before any alarming symptoms make their appearance. It is true that a patient so treated may not sleep more than half as many hours in a given week as when medicinal hypnotics are used, but if signs of exhaustion do not make their appearance, his advance towards recovery will, in my opinion, be more rapid and certain. In asylum practice the routine administration of medicinal hypnotics and "quieting draughts" is reprehensible.

The almost specific effect of opium in certain cases of melancholia I have already spoken of. Of course, the tendency to constipation and loss of appetite which the drug often causes has to be reckoned with and frequently forbids its continued use, notwithstanding the employment of all available counteracting remedies.

The insomnia attending recurrences or exacerbations of excitement, occurring in the course of chronic cases, is generally less pronounced than in primary attacks of acute insanity, but is to be managed in the same way. In chronic melancholia any of the remedies already

recommended, variously combined, may be employed; but measures calculated to relieve the mind, such as a judicious mingling of occupation, diversion, and change of scene, are of most importance.

Delirium tremens may be regarded as a special form of acute insanity in which insomnia is the most prominent symptom; indeed, with few exceptions, the efficacy of remedies in this disease is estimated mainly by their success in inducing sleep. In a fair majority of typical cases, if there be no medication, there will be absolute insomnia with more or less excitement for from three to five days, at the end of which time the patient falls into a sound sleep lasting from five to ten hours, from which he awakes practically convalescent. If, on the other hand, sleep does not supervene, signs of fatal exhaustion soon make their appearance, and death ends the scene. Excepting in comparatively slight cases, until this natural time for sleep arrives, drugs, except in overwhelming doses, are powerless. This natural tendency of the disease to recovery has led to erroneous conclusions as to the efficacy of various drugs in its treatment. Feeding and judicious restraint are of prime importance till the time for sleep approaches; then the wet pack with a full dose of one or more of the hypnotics will be found useful. For this purpose chloral and bromide have my preference. If this is not successful, wait six or eight hours, and repeat the pack and the medication, to which may be added, if there is great muscular activity, hydrobromate of hyoscine hypodermically. Half-ounce doses of tincture of digitalis were quite the vogue some years since, but though this practice is not attended with great danger, it now has few advocates.

Unrefreshing Sleep.

A considerable number of patients who suffer from neurasthenia sleep well, while many more sleep enough but complain that their sleep is not natural or refreshing; one sleeps too heavy, he thinks; another, while he does not exactly dream, is all night in such a distressing mental state—not awake—that he dreads to go to bed. After due attention has been given to the stomach and bowels, posture, covering, ventilation and temperature of the sleeping-room, general treatment directed to the underlying defective state of health must be relied upon. At least I know of no remedy that can be expected directly to relieve this condition.

Nightmare.

Nearly every one who has reached middle life has had one or more attacks of nightmare which he could almost invariably trace to an

attack of acute indigestion. The most prominent feature of order is the peculiar mental state, which consists of a most dis sense of some great and imminent danger, with utter inabilit cape from it or even to move or speak. Very commonly the i to move is felt to be due to immense pressure applied to the Horrid and vivid dreams are usually associated, and go on t great effort the sleeper is able to move or speak, when the a over. Sometimes the eyes are open, and objects dimly see room seem distorted into monstrous demons a few moments the spell is broken. When, however, these attacks become h occurring, perhaps, several times in a night and several nig week without obvious indiscretion of diet, or when in addi the nocturnal attacks the patient has seizures by day with dis and sometimes momentarily impaired or suspended conscic which, judging from his feelings, he positively identifies w former, the serious attention of the physician is challenged. case has gone this far, actual epilepsy has probably develop it will in general be found highly amenable to the treatment to the latter disease.

In fact all sorts of nocturnal seizures occurring in sleep careful investigation. Epilepsy is sometimes wholly nocturn doubtless, in some instances exists for years until finally it is ered by accident. If a patient awakes with a bitten tongue, ference is easy; but if, without apparent cause, he now ar complains of muscular lameness in the morning, the possib nocturnal epilepsy should be thought of.

Pavor Nocturnus, or Night Terrors.

Neurotic children between the ages of two and ten son awake several hours after retiring, screaming and manifestin of intense terror, and for some minutes they cannot be made to nize their surroundings or to respond to pacific measures. they lapse into sleep again without having shown any signs of nition, or they may awaken and accept assurances of safety. the predisposition exists, the most potent exciting causes ap be those acting on the mind some hours before retiring, as gh ries for instance; but errors of diet, worms, and dentition are p not without influence. The treatment consists in obviating the and in the careful observance of hygienic measures. The pr is entirely favorable.

Enuresis Nocturna.

Enuresis nocturna, or bed-wetting, is another common disc sleep in neurotic children, which sometimes persists till puber

commonly occurs a few hours after the child has gone to sleep, and its causes are by no means obvious. It is probable that in most cases the kidneys are unduly active as they may be in hysteria, and in the sound sleep of childhood the nervous mechanism by which the bladder is emptied operates without waking the child. The efficacy of belladonna in the treatment of the disorder supports to some extent this theory of its cause. Measures calculated to give tone to the nervous system, such as cold sponging, are of prime importance. Withholding liquids several hours, and requiring the child to empty its bladder immediately before retiring, are of course rational measures, and curative, too, so far as they tend to break up a bad habit. After puberty sleep becomes less sound and distention of the bladder wakes the patient. Fear of corporal punishment may render sleep less sound, so that the discomfort arising from an overfilled bladder may arouse the patient; and, indeed, as a practical measure vigorous spanking, applied to the nates, has many advocates. There is occasionally a child of such temperament, however, as would be injured by this procedure, but in a large percentage of cases it works well if administered so that the full psychical effect is obtained.

Sleep-Drunkenness or Somnolentia.

Occasionally on waking, persons have been known to have a transient attack of maniacal frenzy. Such attacks have been designated sleep drunkenness or somnolentia, and though very rare they are not without some medico-legal significance, inasmuch as cases have been recorded in which, while in this state, individuals have committed homicide, influenced by a powerful and vivid hallucination. So far as appears, the disorder is confined to sound sleepers when they are suddenly aroused by unexpected circumstances, and they usually remember quite distinctly the hallucination under which they acted. The seizures rarely exceed a minute or two in duration.

Somnambulism.

Somnambulism is a subject which invites the recitation of an almost endless array of curious and more or less interesting facts or statements, yet definite knowledge as to its cause or significance is very meagre. Nowhere can the trite statement more fairly be applied than here, that where fact is feeble, theory is luxuriant. Regarding the theory of somnambulism, I will add nothing to what I have already said under the heading of general considerations. As a disorder of sleep somnambulism will not occupy a very large proportion of the time of the practising physician, because the affection is compara-

tively rare. Most cases occur between the ages of ten and twenty-five and about equally in the two sexes. The affection probably oftener occurs in subjects of the neurotic temperament than in others, but it is certainly met with in individuals who, apart from this, show no evidence of that temperament. In some instances it appears to be hereditary. Among exciting causes mental excitement must be reckoned first, both in children and adults; digestive disturbances, too, claim attention in this connection. Imitation may be a factor, as the disorder has been known to become endemic in schools. In many cases there is no obvious predisposing or exciting cause.

Somnambulism, if made to comprehend all coördinated movements of the voluntary muscles occurring during sleep, accompanied by a variable amount of unconscious ideation, would include sleep talking, which is of course exceedingly common. The term, as ordinarily used, implies that the sleeper rises from bed at least and walks about the room. It is true that he may do certain things that he would not attempt when awake, as walking along the edge of a roof or precipice, and he may play a musical instrument or sing better than when awake; but it is probably not true that he can see better than when awake, and it is certainly not true that he may not receive a fatal fall or suffer any other accident which under the same circumstances he would be exposed to when awake. While in this state it is very difficult to wake the subject, and barring accident, he generally returns to bed before waking.

Suggestion has yielded the best results as a means of treatment. Assure the somnambulist that he will be watched, and if caught in the act, he will surely get a sound rawhiding. In many cases the threat alone, if the patient fully believes it would be carried out, cures the disorder; if, however, the chastisement is once thoroughly applied, occasion for a second recourse to it will seldom be demanded.

Drowsiness.

An undue tendency to somnolence is a common symptom of many disordered states of health, and may more properly be described under the heading of the various diseases in which it occurs than here; but this tendency sometimes shows itself when, in the present state of knowledge, no derangement of the system can be discovered which might account for it, hence nothing remains but to designate it as functional. So designated it may properly be referred to in this article.

Many people after a hearty meal, unless the conditions for it are highly unfavorable, fall asleep for from a few minutes to a half-hour,

and seem greatly refreshed thereby. They present no evidence of disease and claim that these "forty winks" of sleep are almost indispensable to them. Others feel an almost irresistible tendency to sleep under certain circumstances, as at church for example. In the main these must be regarded as instances of habit.

A sudden and short attack of somnolence may occur by itself as an instance of petit mal in idiopathic epilepsy. Such attacks are sometimes termed narcolepsy. They may occur as an early symptom in general diseases, as in the case of a gentleman of thirty-five who ten years before had had a hard chancre and two years before had married a vigorous girl of twenty. He soon became irritable and would fall asleep in spite of himself while participating in a social game of cards. A few months later he had a maniacal outbreak, with thick and tremulous speech, and in a year and a half afterwards died of general paralysis of the insane. This manifestation has been observed also in otherwise healthy people, either associated with such a condition as obesity or without any other apparent deviation from health whatever. It should, if absolutely beyond the control of the patient's will, be regarded as highly suggestive of epilepsy.

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